RESPONSE OF LACTATING DAIRY COWS TO HEAT STRESS, RUMINAL BUFFERS AND VARYING LEVELS OF DIETARY SODIUM AND POTASSIUM: PRODUCTION, DIGESTIVE FUNCTION, ACID-BASE AND MINERAL STATUS

BY

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A DISSERTATION PRESENTED TO THE GRADUATE SCHOOL
OF THE UNIVERSITY OF FLORIDA IN
PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR THE DEGREE OF DOCTOR OF PHILOSOPHY

UNIVERSITY OF FLORIDA

1986

This dissertation is dedicated to

my son, Jordan.

ACKNOWLEDGMENTS

The author extends his gratitude to Dr. David K. Beede for his enthusiasm and concern throughout this program. His devotion to his vocation was consistently evident and this author hopes to maintain a long professional and personal relationship with him.

Special thanks go to Dr. Charles Wilcox, Dr. Robert Collier and Dr. William Thatcher for their professional help, advice and personal interest.

Without the help of fellow students, Quim, Marlin, Jeff, Louis, Chuck, Larry, Francisco, Chik, Paul, Anita, Deanne, Lokinga, John, Bob, Jose, Humberto, Mike, Wendy and Candie Sue, much of this research would have been impossible, especially the fun-filled 26-hour bleeds.

The author also thanks Estelle for her extra effort, and Austin Green and the farm crew for their assistance.

The author will remember always the special friendship extended to him by Quim and Sharon, and Larry and Christina.

The author extends love to Shirley, Mickey, Betsy, Debbie and Yehuda.

TABLE OF CONTENTS

Abstract of Dissertation Presented to the Graduate School
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May, 1986

Chairman: David K. Beede
Major Department: Animal Science

Four experiments were conducted to study responses of lactating dairy cows to heat stress and dietary buffer and mineral levels. Experiments 1 and 2 were designed to evaluate effects of ruminal buffers ($NaHCO_3$ and $KHCO_3$) and varying levels of dietary sodium (Na) and potassium (K) on lactating cows (18 in experiment 1 and 24 in experiment 2) in a heat stress (no shade, NS) or in a control (shade, S) environment. In both experiments, feed intake was lower in NS than S; milk production was lower in NS in experiment 1 but not experiment 2. In experiment 1, 1.0% $KHCO_3$ lowered feed intake and milk yield. Milk yield was higher with 1.5% versus 1.0% dietary K. Milk yield and feed intake were both higher with .85% $NaHCO_3$ versus 0%. In experiment 2, feeding 1.0% $NaHCO_3$ increased milk yield and percent milk fat. Supplementation with .73% NaCl increased milk yield, 1.8% versus 1.3% K increased feed intake and milk yield.

Increasing dietary Na from .18 to .55% with either 1.0% $NaHCO_3$ or .73% NaCl raised 4% fat-corrected milk yield, but together (.88% dietary Na) showed no benefit over .55%.

In experiment 3, circadian patterns of respiration rates, rectal temperatures and blood gas composition between cows in natural (n=12) versus artificial chamber (n=4) environments were similar, confirming that chamber could simulate natural heat stress. Cows in heat stress exhibited signs of respiratory alkalosis during hot daytime hours compared to cows in thermoneutral environment. Turnover rates of liquid digesta (measured by chromium disappearance from rumen) and solid digesta (measured by appearance of ytterbium-marked fiber in feces) from rumen and total volatile fatty acids (VFA) were lower in chamber heat stress than in thermoneutral.

In experiment 4, cows (n=4) heat stressed in chambers and fed high mineral diet (1.25% NaCl and 1.85% KCl added to basal diet) excreted more K in urine but did not exhibit alkalogenic effects on acid-base status resulting from high cation level compared to cows on basal diet. Lower urine pH and higher urine NH_4^+ excretion during cool hours indicated that chloride content of high mineral diet had an acidogenic effect. Ruminal chromium disappearance and VFA fermentation patterns indicated higher liquid turnover rate on high mineral diet, but no effect on solid turnover rate.

CHAPTER I
INTRODUCTION AND REVIEW OF LITERATURE

Introduction

According to United Nations projections, warm climate regions of the world between 30 N and 30 S latitudes will have a population of over 5 billion by the end of this century (McDowell, 1972). Increased production of food from animals will be required to meet future needs. Animal foods are excellent sources of protein. Ruminants play an important role in the food chain because they can utilize plant materials that cannot be eaten directly by humans. Ruminants can consume forage on land areas not ideal for production of cereal grains and utilize dietary nonprotein nitrogen and by-products not suitable for human consumption. Unfortunately, high environmental temperatures and humidity have marked negative effects on grazing behavior, food and water consumption, milk production, milk and blood composition, maintenance requirements, cardio-respiratory function, heat production and body temperature of cattle (Thatcher and Collier, 1981).

Critical temperature is defined as the lowest or highest temperature at which an animal can maintain its body temperature at the basal metabolic rate (NRC, 1981). As ambient temperature increases above the upper critical temperature, the dairy cow must employ certain strategies to maintain body temperature. The upper

critical temperature of dairy animals changes with age, insulation and level of milk production (NRC, 1981).

During thermal stress, alteration in acid-base balance occurs (Dale and Brody, 1954). As ambient temperature increases, respiratory frequency of cattle rises to increase evaporative heat loss from upper respiratory passages. This subsequently can alter blood pH, pO_2 and pCO_2. Carbon dioxide is eliminated faster than it is produced, pCO_2 is lowered, blood pH may rise and the kidney increases excretion of HCO_3 to compensate for the respiratory alkalosis. One might hypothesize that loss of bicarbonate substrate by increased ventilation and urinary excretion would reduce the pool available for salivary buffers that maintain ruminal pH. Additionally, high producing dairy cows are nutritionally challenged to achieve their genetic potential in milk production by feeding diets high in digestible energy. High concentrate rations are known to reduce ruminal pH. Thus usage of dietary buffers may be warranted to aid in maintenance of ruminal pH.

Lactating dairy cows may have a higher dietary requirement for potassium compared to other domestic animals. Reasons include (1) "lactational stress" associated with higher production and high potassium content of milk, (2) heat stress which increases loss of endogenous potassium through sweating (Singh and Newton, 1978) and decreases daily potassium intake and (3) dairy rations which may contain considerable proportions of low potassium by-product feeds, cereal concentrates and other feedstuffs containing low levels of potassium. Voluntary feed intake and intake of required nutrients

decrease as heat load increases above 27°C black globe temperature. As sodium and potassium closely interact in many of their physiological functions, with Na being primarily an extracellular ion and K being an intracellular ion, an increased potassium requirement might also necessitate an increased need for sodium. Earlier research at the University of Florida (Mallonee et al., 1985) demonstrated higher feed intake and milk production with higher than NRC (1978) recommended levels of dietary sodium and potassium during heat stress.

Digestive kinetics are altered during heat stress as nutrient digestibilities may increase and ruminal volatile fatty acid concentrations may decrease (NRC, 1981). Slower rates of passage of digesta through the rumen have been hypothesized to account for these changes (Wayman et al., 1962). Research with cattle within the zone of thermal neutrality has shown that feeding higher levels of NaCl or salts of artificial saliva increases rate of passage and in some cases increases feed intake.

The objectives of this research were

(1) to evaluate effects of heat stress and ruminal buffers ($NaHCO_3$ and $KHCO_3$) on production responses and acid-base status of lactating dairy cows;

(2) to evaluate effects of source and quantity of sodium and potassium on production responses, acid-base status and mineral metabolism of lactating dairy cows during heat stress;

(3) to determine whether acid-base balance, mineral metabolism and rate of passage of digesta through the rumen in lactating dairy cows experiencing heat stress within a nycterohemeral time frame differs from cows not experiencing heat stress; and

(4) to study whether there are differences in ruminal liquid and solid turnover rates, acid-base status and mineral metabolism of lactating dairy cows consuming either a basal or high mineral diet during heat stress.

Functions of Sodium and Potassium

Electrolytes are either anions or cations depending on whether they have a positive or negative charge. They are essential components of all living matter and include the major electrolytes Na^+, K^+, Cl^-, HCO_3^-, HPO_4^-, Ca^{2+} and Mg^+ as well as trace elements Fe^{2+}, Cu^+, Mn^{2+}, Co^{2+}, Cr^{3+}, Cd^{2+}, Zn^{2+}, Br^- and I^- (Tietz, 1982a). Major electrolytes occur mainly as free ions while trace elements are usually associated with proteins. Dietary requirements for individual electrolytes vary, most are needed in small amounts and some like Na, K, P, and Ca must be consumed regularly.

There are almost no metabolic processes which are not dependent on or affected by electrolytes. Functions include maintenance of osmotic pressure between ICF and ECF, water balance, pH, regulation of heart and muscle function, electron transfer and enzyme functions (Tietz, 1982a).

Sodium (Na)

The body is about .2% Na. Roughly 45% is in extracellular fluid, another 45% is in a relatively inert form in the skeleton and the remainder is intracellular (Cunha, 1983a). Most Na is readily exchangeable with the ECF; although about half of the Na in bone is contained on hydroxyapatite crystals deep in long bones. This Na is not osmotically active but may be mobilized during extracellular fluid dilution (Edelman and Leibman, 1959). Plasma Na concentration is about 142 meq/liter (McAdam and O'Dell, 1982). Major functions include regulation of osmotic gradient across cell membrane, acid-base balance, maintenance of cellular membrane potentials, transmission of nerve impulses and involvement in the absorptive processes of monosaccharides, amino acids, pyrimidines and bile salts.

Sodium ion is quantitatively the most abundant cation of the extracellular fluid; it makes up 90% of the osmotically active bases (Houpt, 1982). Its role in osmotic pressure regulation can be approximated as follows (in mmol/liter):

$$Na^{+}(155) + K^{+}(5) + Ca^{2+}(5) + Mg^{2+}(3)$$
$$= Cl^{-}(105) + HCO_3^{-}(30) + \text{proteins } (18) + \text{other } (15).$$

Changes in osmotic pressure largely depend upon Na concentration. Major loss of Na can lead to significant decrease in osmotic pressure and dehydration. Concentration of Na is maintained by regulation of intake and excretion. Little is known about the

mechanism of salt appetite, but many animals are able to control ingestion to just replace a deficiency (Houpt, 1982).

A most important factor in the fate of Na is the amount consumed (Lomba et al., 1969). High intake of Na increases the amount of Na in the feces and Na balance. Sodium requirements for milk do not influence Na digestibility, but do affect the amount of urinary Na. Sodium is absorbed almost completely from the gastrointestinal tract, mostly in the small intestine, and is circulated throughout the entire body (Cunha, 1983a). Excess Na is excreted by the kidneys which are the ultimate regulators of body Na content (Vander, 1980; Poe et al., 1985). Up to 99% of Na filtered by the glomerulus may be reabsorbed in the proximal and distal tubules of the kidney. Reabsorption is controlled partially by adrenal cortical hormones, mainly aldosterone, which increases tubular reabsorption of Na and decreases tubular reabsorption of K (Guyton, 1976).

In ruminant saliva, Na and K ratios increase and decrease according to Na balance. In cattle, which produce up to 100 liters of parotid saliva a day, salivary Na is recycled by reabsorption in the digestive tract (Cunha, 1983a). Polyrrhea during high temperatures can be considerable and can contribute to a negative salt balance (Denton, 1965).

Potassium (K)

Potassium is the third most abundant mineral in the animal body (.3%), after calcium and phosphorus. Major functions are maintenance of acid-base balance and membrane potential of nerves, muscles and other excitable cells, in regulation of osmotic pressure and water

balance, for O_2 and CO_2 transport in blood and as an activator or coenzyme in certain enzyme systems for energy and protein metabolism (Hemken, 1983). Potassium is also a macro-element found in animal tissue and milk. Potassium is present primarily in cells at concentrations of about 150 mmol/liter in tissue cells and 105 mmol/liter in red cells. High intracellular concentration is maintained by active transport mechanism that uses oxidative energy of the cells.

As the main intracellular cation, potassium is required by many enzymes (Ussing, 1960; Lehninger, 1970; Kernan, 1980). Examples are

adenosine triphosphate, which splits phosphate from energy rich ATP and releases energy;

hexokinase, which aids in formation of phosphorylated sugar derivatives in carbohydrate metabolism;

carbonic anhydrase, a decarboxylating enzyme which acts on bicarbonate with CO_2 and H_2O as end products;

cholinesterase, which inactivates muscle stimulation by hydrolyzing acetylcholine to choline and acetic acid; and

galactosidase, which is important for hydrolization of certain polysaccharides to simple sugars in carbohydrate metabolism.

Digestibility of K is 95% or higher with most feedstuffs (Paquay et al., 1969; Pradhan et al., 1974; Grace et al., 1977). The small intestine is the major site of absorption (Pfeffer et al., 1970). As a percentage of the amount of K consumed by sheep, 91.9% entered the small intestine, only 15.1% flowed out and the amount in the feces

was 7.2% of that consumed. Absorption of K increases with increasing K intake (Rogers and Van't Klooster, 1969; Suttle and Field, 1967; Scott, 1967) without altering fecal K (Poe et al., 1985; Greene et al., 1983a; Newton et al., 1972).

Potassium enters the blood by flowing down an electrochemical gradient. The concentration of K in the fluid fraction throughout the gastrointestinal tract is higher than in plasma (Ward, 1966). Absorption of K in the intestines causes only slight and temporary increases in serum level, little moves into the cells and the excess is removed rapidly by the kidneys. This protects against high serum K levels which could cause severe changes in muscle irritability, respiration and myocardial function (Tietz, 1982a). In one study with lactating cows, 75% of K intake was excreted in urine, 13% into the feces and 12% into milk (Ward, 1966). Most of the fecal K was probably of endogenous origin.

Nutrient Interrelationships with Potassium

Effects of K on insulin secretion and blood glucose have been studied (Lentz et al., 1976; Deetz et al., 1981). Increased levels of KCl infused intravenously or intraruminally have been shown to increase insulin. Paquay et al. (1969) have reported correlations between potassium and nitrogen for intake, digestibility, urine and fecal excretion and balance.

Hypomagnesemic tetany is caused by low dietary levels or availability of magnesium (Allcroft and Burns, 1968). Magnesium availability is correlated negatively with level of dietary potassium (Fontenot et al., 1960; Suttle and Field, 1969; Newton et al., 1972;

Greene et al., 1983a, 1983c). Greene et al. (1983b, 1983c) reported that elevated potassium consumption decreases magnesium absorption in preintestinal region. Tracer studies with radioactive magnesium showed that the lower apparent digestibility of magnesium associated with high potassium diets was due to decreased magnesium availability (House and Van Campen, 1970). Martens et al. (1978) suggested that magnesium absorption in the rumen involves an active transport mechanism dependent upon Na^{+}-K^{+}-Mg^{2+}ATPase. DeGroot and Aafjes (1960) reported that high K intake decreased blood magnesium and increased plasma and red blood cell K, but Poe et al. (1985) indicated no effect of high K intake on serum K, Na or magnesium concentrations.

Martens and Rayssiguier (1980) reported that high potassium with low sodium intake was more important than high potassium alone in lowering magnesium absorption. Poe et al. (1985) reported contrary findings. Lambs on high potassium and sodium intakes had increased fecal magnesium and lower magnesium absorption and urinary magnesium.

Newton et al. (1972) reported lower fecal calcium and a trend for higher calcium absorption when a high K diet was fed to sheep. Fontenot et al. (1960) indicated a trend for lower plasma calcium with sheep on high K diet. Negative correlations between calcium and K with cattle have been reported (Paquay et al., 1969; Pradhan et al., 1974; Martens and Rayssiguier, 1980). Poe et al. (1985) found no effect of 2.2% dietary K on calcium metabolism. Erdman et al. (1980) found no effect of dietary calcium from .5 to 1.03% on dietary K requirements with lactating dairy cows.

Fontenot et al. (1960) found increased phosphorus absorption when K increased from 1.45% to 5.08%. St. Omer and Roberts (1967) and Devlin et al. (1969) found higher serum phosphorus with cattle consuming K-adequate rather than K-deficient diets.

Na by K Interactions

Fontenot et al. (1960) and Renkema et al. (1962) reported that addition of dietary Na to ruminant rations has a inhibitory effect on K absorption. Paquay et al. (1969) found no correlations between Na and K since amounts of dietary and urinary Na are so much lower than those of K. Poe et al. (1985) reported no effect of dietary Na on K absorption.

Suttle and Field (1967) added 28 g of K to a hay diet and found increased Na excretion in sheep of 76%. Campbell and Roberts (1965) observed increases in fecal Na as dietary K increased from .36% to .62%. Poe et al. (1983) reported a reduction in Na retention by sheep as dietary K increased from .61% to 3.0%. St. Omer and Roberts (1967) found no effect on fecal Na concentration in beef heifers as dietary K increased from .23% to 1.65%.

Erdman et al. (1980) found no benefit from additional Na (.31 to .52%) to a low (.42%) or adequate (.84%) level of K. Higher Na increased 4% fat-corrected milk at both K levels. Potassium by Na interactions for serum K and Na were significant, indicating that there were interrelationships between the two cations.

Mallonee et al. (1982b) added dietary NaCl at Na levels of .16, .42 and .70% with K levels of 1.07 and 1.58% for lactating cows. Milk yield and feed intake increased with higher Na, but not with

higher K. Response was highest at .42% Na if K is not considered. With K, however, high Na and high K level gave best response suggesting importance of the relative levels of Na and K in lactation rations.

Na and K: Transmission of Electrochemical Impulses

Nerve and muscle cells transmit electrochemical impulses along membranes. In the nerve, transmission depends on separation of K and Na between intracellular and extracellular fluid along the axon (Guyton, 1976). The resting nerve membrane is 50-100 times more permeable to K than Na. Sodium is actively pumped out, K actively pumped in. These two elements, together with large number of poorly diffusable anions are primarily responsible for electrical potential which exists in nerve cells, i.e., the inside negative in relation to the outside. During an action potential the permeability of the membrane increases about 5,000 times and Na ions pour into the membrane. The positive charges neutralize the electronegativity inside and create an excess of positive charges making the membrane potential inside positive. After a fraction of a millisecond, Na conductance decreases to normal and K conductance increases. Potassium ions flow out allowing transfer of positive charges to the outside recreating the negativity inside the cell and returning membrane potential to normal.

Calcium influences action potentials. High levels lower membrane permeability to Na and prevent rapid diffusion of Na into the cell. Low levels of Ca increase permeability and excitability of

cells and are related to parturient paresis in the dairy cow (Hemken, 1983).

Na and K: Osmotic Pressure

Sodium and K are essential in maintaining the osmotic equilibrium between extracellular and intracellular fluids. The osmolarity of a solution depends on the sum of all osmotically active substances in the solution. Potassium accounts for only 1.4% of the total osmolar activity of plasma but for 46.3% of osmolar activity of intracellular fluid (Hemken, 1983). Sodium ion is the chief cation of the extracellular fluid making up about 90% of all osmotically active bases. Changes in osmotic pressure largely depend on Na concentration. A major Na loss can decrease osmotic pressure and cause dehydration (Houpt, 1982). The kidney is a major regulator of plasma Na concentration and thus osmotic pressure.

Na and K: Na^+-K^+-ATPase and the Sodium Pump

The major active transport systems of cells are based on the transport of Na (Lehninger, 1973; Guyton, 1976; Sweadner and Goldin, 1980). There are two major types of systems, one that maintains balance of Na, K and water in the cell and another concerned with inward transport of essential organic nutrients like glucose and amino acids.

Fixed within the cell membrane, having a large particle weight and consisting of two or more component protein molecules is the Na^+-K^+-dependent ATPase system (Lehninger, 1973; Sweadner and Goldin, 1980). It hydrolyzes ATP and moves three Na ions out of cells and two K ions into the cell. The free energy decrease that occurs when

ATP undergoes hydrolysis to ADP and phosphate likely is used to cause a configurational change or rotation of the ATPase molecule in the membrane.

A major fraction of the ATP output of cells active in secretion or transport is used for Na-K-ATPase transport (Lehninger, 1973). The epithelial cells of the kidney may use two-thirds of the total ATP output of its mitochondria to pump Na. The brain uses even a larger fraction. The pump maintains the low Na, high K concentration of blood and other body cells.

The active transport system for sugar and amino acids in the epithelium of the small intestine requires high concentrations of Na in the lumen of the small intestine. If Na is absent, glucose and amino acids are not absorbed since nutrient transport requires an inward gradient and transport of Na. It is postulated that there are two components to this transport system. The first component is the Na concentration gradient. Glucose, for example, and Na are bound to the same carrier which moves into a cell along a Na concentration gradient. A second component is a Na pump which actively transports Na out of the cell creating the concentration gradient that causes Na to flow into the cell. Other inwardly directed active transport processes, i.e., amino acids, potassium or other solutes, likely are similar and require Na (Lehninger, 1973).

Na and K: Acid-Base Balance

Hydrogen ion (H^+) concentration is one of the most vigorously regulated variables in the body (Houpt, 1982). Vital limits of pH variation for mammals may range from 7.0 to 7.8, with the normal

range being between 7.36 and 7.44. Although each body fluid compartment has a different composition and movement of ions or other solutes between these compartments is not necessarily free, blood and plasma are used in acid-base studies since they are easily accessible and reflect the composition of the total extra cellular fluid compartment (Tietz and Siggaard-Anderson, 1982).

Hydrogen ion concentrations are maintained by a number of biological buffering mechanisms involving various systems of the body (Hilwig, 1976). The chemical buffering system, which includes bicarbonate, plasma protein, phosphate and hemoglobin, is the quickest to respond. Acids and bases produced by tissue metabolism are neutralized. Respiratory buffering system provides the main route of CO_2 elimination and is of prime importance in maintaining the bicarbonate/carbonic acid ratio. The kidney eliminates nonvolatile acids and bases.

A buffer system consists of a weak acid and its conjugate base. The relationship between pH and the weak acid and conjugate base is given by the Henderson-Hasselbach equation (Masero and Siegel, 1977):

$$pH = pK + \log \underset{\text{(base)}}{([HCO_3]} / \underset{\text{(acid)}}{[.0301 \times pCO_2]}).$$

Normal ratio of concentration of HCO_3 and CO_2 in plasma is 25 (mmol/liter)/(40 mmHg x .03) or (1.25 mmol/liter) = 20/1 (Masoro and Siegel, 1977). The $\log_{10}$ of 20 is 1.3, therefore the equation appears as such: pH = 6.1 + 1.3 = 7.4. Any change in concentration

of either HCO_3 or pCO_2 and their ratio is accompanied by a change in pH. If an acid (i.e., from absorption of exogenous acids produced in the rumen) is added to the bicarbonate buffer system, the H^+ will react with HCO_3 to form CO_2:

$$HCO_3^- + H^+ = H_2CO_3 = CO_2 + H_2O .$$

The bicarbonate/carbonic acid system is the most important buffer of plasma. It is effective due to high concentrations of its components and the fact that CO_2 can be disposed of readily or retained in the lungs. Also the renal tubules can increase or decrease the rate of reabsorption of HCO_3.

Plasma proteins, i.e., albumins and various classes of globulins, provide a broad spectrum of histidine residue buffer pairs with a range of pK' values from about 5.5 to 8.5 (Masoro and Siegel, 1977). They provide a continuous series of buffers throughout the pH region encountered in plasma. Proteins account for 95% of the non-bicarbonate buffering value of plasma (Tietz and Siggaard-Anderson, 1982).

The total concentration of the phosphate buffer system in both plasma and erythrocytes is less than the other major systems (Masoro and Siegel, 1977). Inorganic phosphate accounts for only about 5% of the nonbicarbonate buffering value of plasma. Organic phosphate in the form of 2,3-diphosphoglycerate accounts for about 10% of the nonbicarbonate buffering value of erythrocyte fluid.

Hemoglobin (Hb) is present in red blood cells at high concentrations. The combination of histidine residues and N terminal valine amino residues causes the hemoglobin titration curve to be almost linear in the pH range of 7-8 (Masoro and Siegel, 1977). More than 60% of the buffering capacity of the red blood cells is in the Hb system and more than 30% in the bicarbonate system with the remainder with the organic phosphate buffers. Ninety percent of blood's capacity to buffer carbonic acid is with the Hb system.

Blood is not a single buffering compartment as the pH of red blood cells is 7.2 and the pH of plasma is 7.4 (Masoro and Siegel, 1977). This is due to the fact that erythrocytes continuously produce lactic acid by anaerobic glycolysis and many ions such as plasma protein polyanions, hemoglobin polyanions, potassium and sodium cannot cross the plasma membrane causing a Donnan equilibrium system to play a role in distribution of diffusable ions like H^+. Nevertheless, due to the rapid interaction of red blood cells and plasma, blood as a whole usually is considered as part of the extracellular buffering system (Tietz and Siggaard-Anderson, 1982).

The bicarbonate system is the major buffer of the interstitial fluid (Davenport, 1974). Proteins and phosphate buffering systems have a very small role. The total volume of interstitial fluid is 3.5 to 4 times that of plasma, so the capacity of interstitial fluid to buffer acids is great.

In normal situations, bone is not significantly involved in acid-base homeostasis (Masoro and Siegel, 1977). In chronic acidotic

states, bone salts accept H^+ and may serve as buffers. Acceptance of H^+ may solubilize bone minerals.

Respiratory buffering mechanism is regulated by the medullary respiratory center which is stimulated by central chemoreceptors located on the anterior surface of the medulla oblongata and by the peripheral chemoreceptors including the carotid bodies and aortic bodies. These receptors are stimulated by a fall in pH of arterial blood or cerebrospinal fluid as well as other factors such as pO_2, exercise and temperature (Tietz and Siggaard-Anderson, 1982).

If acid is added to body fluids, the chemical buffering system reacts first with the formation of CO_2 and the depletion of HCO_3. Ratio of HCO_3/pCO_2 falls and the pH falls slightly. Respiration is stimulated and CO_2 is exhaled. Partial pressure of CO_2 decreases to below normal levels, but the ratio and blood pH are returned toward their normal values. Although the ratio between HCO_3 and pCO_2 is normal, amounts of each are subnormal and full correction of the acid-base abnormality is affected by renal excretion of H^+ and production of HCO_3 (Houpt, 1982).

Na and K: The Kidneys

The function of the kidney is to maintain the constancy of the internal environment (Davenport, 1974). Normal electrolyte patterns of plasma and interstitial fluid are maintained by renal excretion of individual ions. By regulating acidity of the urine and rate of excretion of electrolytes, the kidney helps to keep the pH of plasma within normal limits.

Urine can be either acid or alkaline. When it is acid, the excreted acid is removed from the blood and an equal quantity of base is added to the blood. The capacity of renal tubules to secrete H^+ into the lumen of the tubule is fundamental to HCO_3 reabsorption, titratable acid and ammonia excretion (Hilwig, 1976).

The basic mechanism of H^+ secretion is that a H^+ is derived from carbonic acid formed from CO_2 (Houpt, 1982). The presence of carbonic anhydrase insures a sufficiently rapid formation of carbonic acid. The rate of H^+ secretion by the renal tubules is largely determined by intracellular pH. High pH depresses H^+ secretion into the tubule lumen and a low intracellular pH increases the rate. Intracellular pH generally changes as blood pH or pCO_2 changes. Intracellular K^+ concentration also affects acid secretion. Potassium ions entering the cells displace H^+ ions as the balancing cation for the anionic sites on protein and other organic molecules (Masoro and Siegel, 1977). Release of H^+ by cells may be sufficient to lower pH of extracellular fluid since most of the cells of the body are involved.

During K depletion, a paradoxical situation exists where the blood tends to be alkalotic, but the urine acidic. Due to the reciprocal relationship between H^+ and K^+ ions in the cell, K depletion causes a decrease in renal cell pH which stimulates H^+ secretion thus acidifying the urine. Filtered HCO_3 is not secreted by the kidney but reabsorbed, and more HCO_3 is generated as H^+ is secreted causing an increased alkalotic stress (Vander, 1980; Masoro and Siegel, 1977).

Most excreted H^+ is bound by the bases $HPO_4^=$ and NH_3. As phosphate binds H^+ to form H_2PO_4, Na^+, the cation which electrically balances $HPO_4^=$, is exchanged with the secreted H^+ and returned to the blood.

The base NH_3 is formed by renal tubular cells from glutamine and other amino acids (Vander, 1980; Houpt, 1982). During an acidotic challenge, glutamine catabolism occurring in the liver is shifted to the kidney where amide-nitrogen is excreted in the urine to neutralize and excrete H ions (Trenkle, 1979). Ammonia diffuses along its concentration gradient into the tubular lumen. If H^+ is present, NH_4^+ is formed which is trapped in the tubular fluid and NH_3 continues to diffuse into the lumen.

Filtered bicarbonate is reabsorbed from tubular fluid via the same mechanism that secretes H^+ into tubular fluid (Masoro and Siegel, 1977; Davenport, 1974). For every HCO_3 removed from tubular fluid and thus from plasma by reaction with H^+, a HCO_3 is added to plasma. All HCO_3 will be reabsorbed if H^+ secretion rate exceeds HCO_3^- filtration rate. If H^+ secretion rate is less, HCO_3^- will appear in the urine. Under a base stress, i.e., respiratory alkalosis, less H^+ is secreted, more HCO_3 appears in the urine and the urine will be alkaline.

Ruminal fermentation provides a constant tendency to metabolic acidosis (Kronfeld, 1976). This tendency is lower on high roughage diets and higher on high concentrate diets. After a meal, there is an acid tide in the urine due to secretion of HCO_3 in the saliva and production of volatile fatty acids. Volatile fatty acids are

absorbed through the rumen undissociated, they dissociate upon entering the blood which diminishes blood HCO_3 and tends to diminish blood pH.

Scott (1971a, 1972) fed calves high roughage or high concentrate diets with and without infusion of HCl into the rumen. On the high roughage diet, urine was alkaline due to excretion of HCO_3. Hydrochloride acid infusion lowered urinary pH due to acid excretion of which about 20% was H_2PO_4 and about 80% was NH_4^+. On the high concentrate diet, urine was acidic and HCl infusion increased secretion of NH_4^+.

In cows during heat stress, increased respiration is a strategy to dissipate heat (Bianca, 1965). This causes excessive elimination of CO_2 and an increase in the HCO_3/pCO_2 ratio which may be associated with an increase in blood pH. The compensatory mechanisms of the body to the respiratory alkalosis are functions mainly of the kidney which responds by decreased H^+ secretion, decreased formation of NH_3 and decreased HCO_3 reabsorption.

The kidney is the most important regulator of body Na and water. Sodium and water are freely filterable at the glomerulus and undergo more than 99% tubular reabsorption, but no secretion. Sodium reabsorption is an active process in that it is carrier-mediated, requires an energy supply and can occur against an electrochemical gradient. Active tubular Na reabsorption is the primary force which results in reabsorption of chloride and water. Only in the loop of Henle is Na reabsorption dependent on active chloride reabsorption (Vander, 1980).

In the proximal tubules of the kidney, Na^+ enters the cell by facilitated diffusion along an electrochemical gradient created by Na-K-ATPase dependent active transport of Na across the basolateral membrane into the intercellular spaces. Removal of Na from the lumen lowers total lumen osmolality and raises osmolality in intercellular spaces causing net diffusion of water into intercellular spaces.

The potassium concentration of the extracellular fluid is controlled by the kidney. Potassium is completely filtered at the glomerulus (Vander, 1980). Potassium is also reabsorbed and secreted by the tubules. Reabsorption is by active transport and occurs at the proximal tubule, ascending loop of Henle, distal tubules and collecting duct. Final secretion of K by the nephron may be much more or less than was filtered.

Secretion of K involves active transport into the cell at the basolateral membrane coupled to Na via the Na-K-ATPase system and passive exit at the luminal membrane. This passive luminal step depends on intracellular K concentration, luminal concentration, the potential difference opposing diffusion into the lumen and the permeability of the luminal membrane to K (Guyton, 1976; Vander, 1980).

Ruminants evolved as consumers of large amounts of potassium and thus much of the dietary potassium intake is excreted into the urine. Potassium secretion into the tubular fluid in the distal tubule and collecting duct, along with HCO_3, results in an alkaline urine (Hilwig, 1976). Potassium secretion can also occur with an acid urine.

Antidiuretic hormone (ADH) is the major determinant of water permeability in the late distal tubule and the entire cortical and medullary collecting duct system. Release of ADH is caused by an increase in plasma osmolality (excess Na) which excites osmoreceptors in the supraoptic nuclei of the hypothalamus. ADH increases permeability of the collection tubes to water (Guyton, 1976).

Aldosterone increases activity of Na retaining process including uptake of Na from the gastrointestinal tract and reabsorption of Na from renal tubules in exchange for K^+ or H^+ ions (Gans and Mercer, 1982; Dickson, 1982). Aldosterone secretion is controlled by the kidney. As decreases in blood pressure and volume or Na concentration are detected, juxtaglomerular apparatus of the kidney secretes renin, an enzyme, which cleaves the decapeptide angiotensin I from angiotensiogen in the plasma. A converting enzyme most abundant in pulmonic circulation cleaves a dipeptide from angiotensin I forming angiotensin II. Angiotensin II is potentiated by ACTH and high plasma K and causes secretion of aldosterone by zona glomerulosa cells of the adrenal gland cortex. Aldosterone exerts its effects on sweat glands, salivary glands, intestinal mucosa and distal convoluted tubules of the kidney promoting Na absorption and retention (Coppock and Fettman, 1978; Guyton, 1976; Dickson, 1982).

An increase in K concentration causes an increase in aldosterone concentration which then causes increased excretion of K by the kidneys (Guyton, 1976). Absorption of Na from the tubules causes an electronegativity in the tubules. This electrical gradient attracts K^+ from extracellular fluid of the kidneys into the tubules. If for

any reason too little Na is available for exchange with K, K secretion may become deficient.

Na and K: Saliva

Ruminants secrete enormous quantities of saliva. Saliva moistens and lubricates food and assists in mastication and swallowing. It contributes more than 70% of the water and most of the salts to the rumen. Bicarbonate and phosphate buffers contained in saliva help maintain rumen pH (Bartley, 1976). Saliva contributes to maintenance of the inorganic composition and fluid volume of rumen contents (Baily, 1961a). Feed and water intake as well as nutrient turnover rates are influenced by saliva. Nitrogenous and mineral nutrients for microorganisms are supplied by saliva. Sodium and potassium constitute about 90% of cations of saliva (McDougall, 1948). The 24-hour saliva production of a 700 kg cow consuming hay and grain may reach 190 kg (Bartley, 1975). Of this, $NaHCO_3$ may make up 1100 g, Na_2HPO_4 350 g, NaCl 100 g and nitrogen and urea.

Parotid saliva normally contains about 18 times more Na than K. Denton (1956) reported that when sheep with parotid fistula were deprived of Na, salivary Na fell from 178 to 46 mEq/liter and K rose from 21 to 140 mEq/liter. The tonicity of the fluid was unchanged. An increase or decrease in Na content of mixed saliva is accompanied by a commensurate increase or decrease of K (Baily and Balch, 1961b). This is due to aldosterone secretion from the stress of Na depletion.

Saliva secretion during feeding may cause a slight hemoconcentration which, along with increased osmotic pressure from

nutrients absorbed from the gut, could stimulate release of antidiuretic hormone (Scott, 1975). The ruminant can also retrieve products of salivation as the kidney conserves both Na and water (Stacy and Warner, 1966).

With increased salivary secretion, K and phosphate concentrations in saliva tend to decrease, Na and bicarbonate concentrations tend to increase (Baily and Balch, 1961). They found that Na concentration in rumen contents was parallel to, but lower than concentration in saliva. This suggests that as food was eaten and salivary flow increased, absorption of Na and voluntary water intake was sufficient to prevent any rise in Na concentration.

Ruminal K concentration reflects differences in amounts of K supplied by saliva, these concentrations are higher than those found in either saliva or plasma. High K concentration found after a meal is due to a rapid solution of dietary K in rumen water, and the subsequent decline is due to absorption from the rumen and dilution by saliva (Baily and Balch, 1961; Warner and Stacy, 1972b).

The ionic composition of ruminal fluid is closely related to rate of salivary secretion and concentrations are comparable. Potassium values in saliva range from 4 to 70 mEq/liter and in rumen fluid from 24 to 85 mEq/liter. Sodium concentrations in saliva range from 74 to 166 mEq/liter and from 83 to 147 mEq/liter in ruminal fluid (Baily, 1961a and b). Ruminal potassium concentrations reflect potassium content of diet, however. In contrast, more Na is present in rumen fluid than what is usually in the diet, indicating the importance of saliva as the main source of Na in the rumen.

Na and K: Rumen

Sodium and potassium are required by ruminal microorganisms (McLeod and Snell, 1948; Hubbert et al., 1958; Bryant et al., 1959; Caldwell and Hudson, 1974). Most predominant rumen bacteria contain enzymes that require K (Durand and Kawashima, 1980). In an in vitro study with different species of ruminal bacteria, Caldwell and Hudson (1974) found optimal growth at sodium concentrations similar to that of ruminal fluid (60 to 120 mM). Maximum growth for Bacteroides succinogenes was attained at 3-10 mM K (Bryant et al., 1959). Bacteroides amylophylus reached maximum growth at K concentrations of 1.2 mM (Caldwell and Hudson, 1974). Caldwell and Hudson (1974) also found significant Na x K interaction: at low Na levels higher K levels were necessary for maximum growth. Durand and Kawashima (1980) found 12.8 to 38.5 mM of available K is required for optimal fermentation, but it was not established how much was for microorganisms and how much for external physiochemical factors. Potassium is more important for protein synthesis than glycolysis and has been shown to be associated with ribosomes of Aerobacter aerogenes.

Minerals contribute more to rumen osmolality than other solutes including VFA's, except postprandially with concentrate feeding (Bennink et al., 1978). A large addition of minerals to a diet may decrease fermentation by increasing ruminal osmotic pressure.

Ion transport across ruminal epithelium is affected by state of nutrition. Scott (1965) found that a readily metabolizable source of energy was necessary for the maintenance of a normal potential

difference across the wall of temporarily isolated loop of small intestine in sheep. Furthermore, Warner and Stacy (1972) concluded that since Na is the predominant cation in the rumen and since there is no evidence for any appreciable compensatory osmotically active substances, absorption of Na from the rumen is a major mechanism maintaining the resting rumen contents hypotonic to blood. Volatile fatty acid absorption plays a large part, but not in the resting rumen. In the rumen epithelium, Na-K-ATPase is likely involved. Sodium absorption from the rumen may be due to effect of increased osmotic pressure on the activity of key enzymes involved in the transport of the ion across cell membranes (Warner and Stacy, 1972). Sodium is absorbed against a concentration gradient and electrical potential due to this active process in the epithelium (Dobson, 1959). Potassium may be actively secreted into the rumen and K concentrations are larger than plasma concentrations (Ferreira et al., 1966).

Sodium and K concentrations both affect rate of Na absorption, but K concentration in rumen fluid may directly stimulate the amount of Na absorbed (Scott, 1975; Warner and Stacy, 1972). High K diets fed to sheep increased K to Na ratio in the rumen (MacGregor and Armstrong, 1979). The fate of the absorbed Na is determined by the kidney, which is also influenced by K. In order to maintain the ionic composition of the rumen when dietary K is reduced, absorption of Na from the rumen decreases and Na accumulates in the rumen. A temporary degree of extracellular Na depletion may occur. Urinary excretion of Na will be reduced likely as a result of the release of

aldosterone (Scott and Dobson, 1965). This suggests that the requirements of the rumen dominate response of the whole animal to dietary changes in electrolyte intake (Warner and Stacy, 1972). Adjustments within the rumen may lead to disturbances of the animal's overall electrolyte balance.

In the omasum of sheep and goats, about 25% of the Na and a little less than 10% of K that entered from the reticulum was found to be absorbed (Kay and Pfeffer, 1970).

Na and K: Small and Large Intestines

Sodium and potassium, the principal cations throughout the gut, are maintained at fairly constant ionic concentrations throughout the small intestine of sheep (Kay and Pfeffer, 1970). As digesta pass down the small intestine, concentrations of Na and K fluctuate in a reciprocal manner; the concentration of Na can be doubled and that of K halved. This is partly due to bile, pancreatic juice and other intestinal secretions which have high Na concentrations and low K concentrations.

There is little net absorption of Na from the small intestine of sheep, but the large intestine absorbs Na very efficiently. This is reversed for K: the small intestine absorbs most of the K entering but little absorption takes place in the large intestine and K concentration increases as water is absorbed. In cattle, Na concentration decreases in the large intestine as in sheep, but there is no corresponding increase in K. Fecal water of cattle is hypotonic to plasma whereas in sheep fecal water is hypertonic (Kay and Pfeffer, 1970).

Sodium ion transfer across mucosal membrane of intestines is an active process, involving Na-K-ATPase that requires the presence of both cations (Allen, 1982).

Two major variable constituents of abomasal secretions are H^+ and Na^+ (Phillipson, 1982). Harrison (1962) reported that in sheep the flow of bile is about 20 to 40 ml/hour with cation concentrations of about 154 mEq/liter for Na and 6.7 mEq/liter for K. The volume of pancreatic juice secreted by cattle is between 2.2 and 4.8 liter/day and by sheep .32 to .42 liter (Phillipson, 1982). The concentration of Na in the pancreatic juice of sheep is 135 to 165 mEq/liter and of K about 3.9 to 5.4 mEq/liter (Taylor, 1962). In the small intestine of sheep, Na concentration is about 135 mEq/liter and K concentration is about 8 mEq/liter.

Na and K: Sweat

Sweating rates for cattle increase as environmental temperature increases. McDowell and Weldy (1967) reported nonlactating cows at 30°C lost 67.9 kg of water with 29.3 kg lost from the body surface compared to 42.9 kg water lost at 20°C with 10.6 kg water lost from the body surface. This suggests a considerable loss of K as K is the most abundant ion in the sweat of ruminants. In another study (Johnson, 1970), animals were exposed for 4 hours to air temperatures of 20-45°C at 30% relative humidity or they were exposed for 5-7 hours at 40 and 45°C at 40% relative humidity. Potassium content of sweat increased as temperature increased above 30°C. Singh and Newton (1978) exposed cattle to 40.5°C at 50% relative humidity for 12 hours a day up to 14 days and found that the K content of sweat

increased with time, suggesting that repeated exposure to high temperature will increase loss of K through the skin. Jenkinson and Mabon (1973) exposed calves to 15, 25, 35 and 40°C temperatures. Calves consumed 52 grams of K per day. Potassium losses via the skin as percentages of dietary intake were estimated to be .20, .99, 2.28 and 11.45%, respectively, as temperature increased. Jenkinson and Mabon (1973) hypothesized from the fact that the Na/K ratio of cattle sweat was low that sweating involves either active secretion or selective reabsorption through the long straight duct of the gland.

Mallonee et al. (1985) collected sweat secretions from acclimated Holstein and Jersey cows in either shade or no shade environments for 2 hour intervals at 0900, 1300 and 2000 hours. During the first sampling time, black globe temperature averaged 31°C in shade and 45°C in no shade environments. Cows without shade had started to sweat and K losses were almost three times greater than K losses of cows in the shade. During the hottest hours of the day, cows without shade experienced black globe temperatures of about 50°C and had increased K loss 300% from earlier. Cows in shade experienced black globe temperature about 37°C and K loss increased about 65%. Losses of K in sweat for both groups were substantially lower at 2000 hours and almost identical between environments.

Na and K: Milk

Electrolyte content of milk is similar to that of intracellular fluid (Schmidt, 1971). The K content is approximately .15% and the Na content is about .06%. Sodium diffuses down a concentration gradient from extracellular fluid, through the epithelial cell of the

mammary gland into the milk where it is actively extruded by Na-K-ATPase from the epithelial cell back to the extracellular fluid. Potassium passively diffuses down a gradient from the cell to the milk and may also be reciprocally transferred with Na via the Na-K-ATPase.

Kamal et al. (1961) reported slight but continuous decline in K content of milk throughout the lactation period, whereas Na increased with stage of lactation. Pradhan et al. (1974) reported cows in early lactation averaged .16% K in milk while cows in mid and late-lactation had about .14 and .12% K, respectively. Most variation in the K content of milk is due to individual variation (Sasser et al., 1966), more than that due to breed, stage of lactation, temperature or other factors.

Consumption of diets severely deficient in K will reduce feed intake, milk yield and the K content of milk compared to cows fed a K-adequate diet (Pradhan and Hemken, 1968; Paquay et al., 1969; Mallonee et al., in press). Cows fed moderately K-deficient diets (.45 or .51% K) did not exhibit significantly different K levels in milk (Dennis and Hemken,1978). Sodium deficient diets will also reduce appetite and milk yield (Cunha, 1983a) although the Na, Cl and K content of milk did not change in experiments cited by Cunha (1983a). Appearance of deficiency symptoms apparently is dependent on the level of milk production.

Na and K: Dietary Tolerances

Signs of Na (and Cl) deficiency in cattle include a salt craving evidenced by licking soil, rocks and other objects (Cunha, 1983; NRC,

1980). There is a loss of appetite and production. Animals appear unthrifty and the hair coat roughens. The end result of long term deficiency is death.

The major factor that influences salt toxicosis is availability of drinking water (NRC, 1980). With an adequate supply, large quantities of dietary salt can be tolerated. The maximum recommended salt level for dairy cows is 4%, that being the maximum amount tested.

Potassium toxicosis is unlikely to occur under practical situations, but high levels may predispose animals to hypomagnesemic tetany (Ward, 1966). Toxicosis may occur from the feeding of excess levels of K supplements. Administration of 393 grams of K as KCl by stomach tube to five cows of about 300 kg resulted in one death and two cows required treatment (Dennis and Harbaugh, 1948). Oral administration of 501 mg K per kg of body weight to a 475 kg dairy cow by stomach tube resulted in death within one minute by cardiac arrest (Ward, 1966). This dose was about half the daily intake of cows fed 15 kg of alfalfa hay/day with no ill effects.

The most general sign associated with K deficiency is a decrease in feed intake (St. Omer and Roberts, 1967; Mallonee et al., 1982a) and reduced milk yield (Dennis and Hemken, 1978; Mallonee et al., 1982a). Pradhan and Hemken (1968) fed lactating cows .26, .15 and .06% K. After 3 weeks, deficiency symptoms were manifest for .15 and .06% K. In two experiments, Mallonee et al. (1982a) fed twelve early and mid-lactation cows an adequate K diet (.96%) for 3 weeks, then eight cows were fed a K deficient diet of .11%. These cows stopped

eating almost immediately, average dry matter intake fell to about .2 kg/day and milk yield dropped 64% by the fifth day. Pica was observed by the second day. This first experiment was terminated on day 6 due to the death of one cow and signs of tetany exhibited by a second. The remaining cows on the deficient diet were given 90 g KCl intraruminally and offered a K adequate diet. All commenced eating within 1 hour.

In the second experiment, ten cows in mid-lactation were subjected to the same experimental design. Feed intake and milk yield declined quickly as in the first experiment. On day 16, all cows were fed K sufficient diets and intake returned to previous adequate levels. One cow died on day 8 of the depletion phase in this experiment. In both experiments, the deaths occurred in higher producing cows as more K was being lost via the milk by these animals. Near complete inanition and pica were observed in these two experiments. Cows could apparently 'detect' absence of presence of K (as Cl salt) in the diet.

Na and K: Feeds and Dietary Requirements

Commonly used feeds do not contain sufficient Na to meet body needs and NaCl is usually supplemented (Cunha, 1983). Sodium requirements may be influenced by temperature and humidity as animals in hot humid environments lose some Na via the sweat. Often forages in these areas are low in Na. The Na content of water influences requirements. Sodium content of some feeds can vary in different areas, seasons and processing methods. Cattle consume more salt when a forage is succulent compared to when it is fed dry or mature. The

stage of the animal's life cycle influences salt needs. The 1978 NRC Nutrient Requirements of Dairy Cattle recommends .18% Na in dietary dry matter. Usually NaCl supplementation in the concentrates is recommended.

Potassium is generally found in adequate amounts in forages, with the exception of corn silage (Hemken, 1983; NRC, 1980). The K content of forages can be affected by stage of maturity of the plant since K decreases as the plant matures, by species, by interspecies competition when grown in mixtures, by management procedures, fertilization and soil environmental conditions.

Dietary K requirements for dairy cattle have been investigated (DuToit et al., 1934; Pradhan and Hemken, 1968; Dennis et al., 1976; Erdman et al., 1979; Beede et al., 1981; Mallonee et al., 1982b). For many years, the study of DuToit et al. (1934) was cited to illustrate that supplemental K was not needed and that .32% K was ample for the production of just over 2 gallons of milk per day. Ward (1966) suggested that dietary requirement was about .5%. Pradham and Hemken (1968) found .8% beneficial over the lower levels. Dennis et al. (1976) reported .45% K was adequate with early lactation cows, but body weight and feed intake were greater with .66% K. In another experiment, Dennis and Hemken (1978) reported increased feed intake and milk yield by increasing dietary K from .46 to .97%.

Most grains cannot supply the K requirement for lactating cows. Oil meals, sugar cane and sugar beet molasses are high in K. By-product feedstuffs such as brewers dried grains, distillers dried

grains and corn gluten meal are low in K as are cotton seed hulls. The K content of most feed ingredients is highly variable and when published values are used to balance diets, the K content should be increased or laboratory analysis should be used.

Thermoregulation

Heat Dissipation During Heat Stress

An animal must lose heat continuously in order to maintain body temperature. Sensitive mechanisms balance changes in heat production with equivalent changes in heat loss. It is generally accepted that the hypothalamus contains centers for the major control of heat balance (McDowell, 1972). In the mammal, a high priority is placed on thermoregulation, to the extent that other functions, i.e., lactation, growth and reproduction, are reduced (Thatcher and Collier, 1981). The zone of thermal neutrality, bounded by upper and lower critical temperatures, is the range of environmental temperatures that a homeotherm can withstand without changes in basal metabolism. Temperature is maintained within this zone by adjustments in blood flow, pelage or behavior (Thatcher and Collier, 1981).

Heat is exchanged with the environment by means of conduction, convection, radiation and evaporation (McDowell, 1972; Thatcher et al., 1981). Evaporation is termed insensible heat loss and does not require a thermal gradient. Conduction, convection and radiation are termed sensible heat loss and do require thermal gradients. Conduction is passage of heat energy from particle to particle due to

a temperature gradient. Convection is transfer of heat energy across space without heating the space through which it passes. Evaporation refers to vaporization of water from body surfaces and respiratory tract (McDowell, 1972). When air temperature is low, conduction of heat from the body is rapid. However, when air temperature is high skin temperature increases and conduction of heat is slowed.

Evaporation is the most efficient means of removing heat from the body and takes place from the respiratory tract and skin surface. Water reaches the skin by simple transudation from underlying tissue, via the sweat glands or by external application. Evaporation is affected by relative humidity and air flow.

With conduction and convection, transfer of heat takes place in whatever direction the gradient indicates, including from hotter air to cooler skin.

Strategies employed by animals during high ambient temperature take place in approximately the following order: (1) changes in vascular blood flow, (2) initiation of sweating, (3) increased respiration rate, (4) changes in endocrine activity, (5) changes in behavior, (6) increased water intake, (7) increased body temperature, (8) changes in use of body water, and (9) changes in state of hydration (McDowell, 1972).

As ambient temperature begins to increase, adjustments in the cardiovascular system occur before the upper critical temperature is reached (Thatcher and Collier, 1981). Adjustments are in cardiac output, blood volume and peripheral vasodilation as more blood is shifted to the skin. Since conduction, convection and radiation

require a gradient for heat to flow down, vasodilation of peripheral blood vessels increase the amount of blood and heat brought to the body surface for dissipation.

In order to move more water to the body surface for heat dissipation, plasma volume is expanded (McDowell, 1972). This can be seen as decreased percent plasma protein and hematocrit. This plasma volume shift can occur quickly (Conley and Nickerson, 1945). Israel et al. (1978) have observed a decline in percent plasma protein as ambient black globe temperature increased reflecting a plasma volume expansion to meet increased surface water requirements.

Respiration During Heat Stress

Increased respiration rate is a mechanism to increase heat dissipation (Bayer et al., 1980). It contributes to dissipation by means of evaporation from the respiratory tract especially the upper respiratory tract (Thompson, 1973). When cattle are exposed to hot environments, respiration rate increases and tidal volume decreases with the net effect of increased respiratory minute volume (Thompson, 1973; Bianca and Findlay, 1962). In addition, increased rate of breathing is linked with increased muscle work and this means added heat production so that additional heat dissipation is small. Accelerated breathing does increase heat dissipation in the region of the head and upper respiratory passage due to a countercurrent heat exchange mechanisms. This is characterized by an artery entwined within veins (Bayer et al., 1980). Heat is returned to the body from the head via venous flow while arterial blood flowing to the brain is cooled. For example, when cattle increase respiration rate during

heat stress, there is increased heat loss from the cerebral arterial blood to venous blood from the nasal and oral mucosal area which prevents the brain from overheating (Baker and Hayward, 1968).

Efficiency of panting depends on humidity of atmospheric air. Expired air is almost saturated with moisture of body temperature, so increased humidity of inspired air will decrease respiratory evaporative heat loss for a given respiratory minute volume. McLean and Calvert (1972) found that increasing the relative humidity of atmospheric air from 32 to 72% at 31°C only slightly decreased respiratory evaporation. This was due to an increase in respiratory frequency which compensated for the increase in relative humidity. In severe heat, i.e., high relative humidity and air temperature where body temperature increases, panting is followed by a 'second phase' of breathing with low respiratory frequency and high tidal volume (Bianca, 1965).

Panting is not as effective as sweating in increasing evaporative moisture loss (Thompson, 1973). This suggests that respiratory activity is not the decisive factor in control of body temperature during heat stress. High respiratory activity during heat stress may be a measure of the inadequacy of the quantitatively more important cutaneous evaporation to maintain thermal balance (Riek and Lee, 1948).

Another factor to be considered is that panting results in increased convection in the animal. If a cow is exposed to temperatures above that of the respiratory evaporative surface, heat is gained (Bayer, 1980).

Panting tends to alter alveolar ventilation which may subsequently alter blood pH, pO_2 and pCO_2. Carbon dioxide is eliminated faster than it is produced, pCO_2 is lowered, the ratio of HCO_3 to pCO_2 is changed and blood pH may rise. To maintain blood pH, HCO_3 is secreted by the kidney as was discussed previously.

Feed Intake and Digestive Physiology During Heat Stress

In thermoneutral temperatures, feed intake does not vary due to temperature, but in higher temperatures intake decreases and in low temperatures intake increases as strategies to maintain body core temperature.

As feed intake decreases, intake of absolute amount of essential nutrients are lowered unless nutrient density of the meal is increased. This decrease in feed intake with rising environmental temperature conforms to the idea that food consumption is at least partly determined by the ability to dissipate the heat generated by the metabolism of food. The hypothalamus may act as an integrator for the regulation of feed intake and other functions involved in energy balance (Brobeck, 1960; Baile and Forbes, 1974). In evidence of this, Andersson and Larsson (1961) warmed the pre-optic area and rostral hypothalamus of goats with thermodes which caused hungry animals to stop eating.

Motility of the reticulo-rumen is regulated by 'gastric centers' located in the medulla oblongata which receive afferent sensory inputs from the forestomach and which influence the activity of the efferent parasympathetic nerves to the forestomach (Leek and Harding, 1975). Examples of estabished afferent fibers are tension receptors

which enhance motility when the rumen is distended and acid-sensitive receptors which reduce motility when exposed to volatile fatty acids or low pH. Ruminal motility responds quickly to cold and heat stress, the rapidity of the response suggests that a neural mechanism is involved (Christopherson and Kennedy, 1983).

Production of ruminal volatile fatty acids is lower during heat stress likely as a result of decreased substrate consumption (Gengler et al., 1970; McDowell, 1972). However, when feed intake was maintained by feeding weighbacks via ruminal fistula, volatile fatty acid production was still lower (Kelly et al., 1967). Dale and Brody (1954) suggested that during heat stress lactating cows might experience ketosis since lowered energy input would result in catabolism of fat, however, they observed neither ketosis nor ketonuria.

Dietary protein utilization and body protein metabolism have been shown to be altered during heat stress (Ames et al., 1980; Kamal and Johnson, 1970; McDowell et al., 1969). Reduced feed intake will result in less dietary protein. Kamal and Johnson (1970) detected 69% decrease in nitrogen retention by cows at 32.2 versus 18.3°C.

There is evidence for reduced blood flow to the ruminant digestive tract during heat stress (Hales, 1973; Von Engelhardt and Hales, 1977). Decreased blood flow might increase the concentration of volatile fatty acids and further decrease motility. Ruminal lactic acid concentration has been shown to be higher and ruminal pH lower during heat stress in cattle (Mishra et al., 1970). It is therefore conceivable that a high lactic acid concentration and low

ruminal pH might be involved in inhibiting ruminal motility during heat stress. Kelly et al. (1967) observed that heat stress caused a depression rather than an increase in ruminal volatile fatty acid concentration which is not consistent with the above theory.

Feed intake also is reduced during heat stress by increased respiration rate and water intake (Beede et al., 1981; Roman-Ponce et al., 1977). In addition to increased water intake, decreased gut motility (Atterby and Johnson, 1968) and rumination lead to gut fill. Rumen contraction rate is also reduced during heat stress (Collier et al., 1981). Reduced rate of passage (Warren et al., 1974) has also been hypothesized to account for decreased feed intake. Longer retention time slightly enhances digestibility of digestible energy and fiber. Observed changes in feed digestibility are not solely dependent on feed intake since the effects are also observed when feed intake is equalized (Lippke, 1975; Warren et al., 1974).

Another more indirect factor which is involved with decreased feed intake is the change in behavior directed toward decreasing heat production, i.e., movement of animal from feeding areas to seek shade or water (McDowell, 1972).

Generally, as the environmental temperature increases up to about 30°C, there is a depression in gross efficiency, a decreased feed intake and lowering of total heat production. Feed efficiency (megacalories of metabolizable energy intake per kilogram of milk) declines rapidly above 27°C (Moody et al., 1967). Above 30°C, heat production increases due to added heat from the increased activity of

heat loss mechanisms. A greater proportion of energy consumed therefore goes for maintenance resulting in a decline in gross efficiency (McDowell, 1972).

In a series of experiments with lactating Holsteins (McDowell, 1972), efficiency of utilization of digestible energy for milk yield was 60% at 21°C, but only 40% after 7 days at 32°C and 31% after 14 days. Average daily consumption of DE decreased about 16% at 32°C, but daily output of milk energy decreased more than 22% indicating that cows consumed more energy than they needed but gave poorer returns.

Thyroid activity decreases during warm temperatures and increased during cold (NRC, 1981). A shift in thyroid activity may be associated with both a change in gut motility and rate of digesta passage. Thyroid hormones have been shown to influence gastrointestinal motility in several species (Levin, 1969) including ruminants (Miller et al., 1974; Kennedy et al., 1977). Kennedy et al. (1977) showed that thyroidectomy of sheep resulted in prolonged retention time and increased digestibilities whereas supplemental or replacement thyroid hormone had the opposite effect.

Milk Production and Composition During Heat Stress

Decreases in milk yield have been reported starting at environmental temperatures of about 27°C (Worstell and Brody, 1953; Regan and Richardson, 1938; Ragsdale et al., 1948). At the University of Florida experiment station, climatic measurements have been related to milk yield and composition (Rodriguez et al., 1977). Relative humidity and maximum and minimum temperatures

accounted for 1-6% of the variability in milk yield. Maximum daily temperature effects on milk yield were slight between 9 and 27°C but milk yield declined above 27°C.

Decline in feed intake is a major factor associated with decrease in milk yield due to heat (Johnson et al., 1966). Maintaining intake via ruminal fistula maintained lactation although full production was not supported. Bandaranayaka and Holmes (1976) controlled feed intake via ruminal fistula in Jersey cows at 30°C and observed higher rectal temperatures and respiration rates, but lower milk yield than cows at 15°C. Brody et al. (1954) reported improved milk yield during heat stress with increasing wind speed up to 16 km/hour at 35°C compared to .8 km/hour at 35°C. Both feed intake and milk yield were improved for cows receiving 20.8% crude protein diets versus 14.3% (Hassan and Roussel, 1975).

Effects of hot environment on stage of lactation were studied by Maust et al. (1972). Cows which calved in early summer produced about 11% less milk than those that calved in winter. Mid-lactation cows were most adversely affected, late and early lactation cows the least. Cows in early lactation, however, catabolized more of their body reserves. Johnson et al. (1960) also reported greater depression in milk yield for cows in early lactation. Furthermore, animals may become acclimated as milk yield of Holsteins was 20% lower and that of Jerseys 8% lower during the first week of exposure to heat stress (Brody et al., 1955). After the first week, the magnitude of depression became smaller.

In Louisiana, Holsteins that calved in the hot season produced 5 to 8% less milk than cows that calved during the rest of the year (Branton et al., 1974). In Florida, cows in no shade lot for the last trimester of pregnancy had calves with lower body weight compared to cows in shade lot (Collier et al., 1982b). Milk yield was correlated in a linear manner with calf birth weight and cows in no shade lot produced less during the subsequent lactation compared to cows in shade lot.

Percent milk fat, solids not fat and total solids are highest during winter and lowest during the summer (Wilcox et al., 1959; Spike and Freeman, 1967). Protein and mineral content are lower in summer. Collier et al. (1981) detected no difference in freezing point depression, percent acidity, total protein, percent fat, somatic cells per ml milk or cases of mastitis for 48 Holstein and Jersey cows in either a shade or no shade management system. Other reports have indicated an increased percent milk fat with lower milk yield during heat stress (Ragsdale et al., 1950; Cobble and Herman, 1951); however, yield of milk fat decreased (Richardson et al., 1961). Most workers agree that the solid not fat content of milk decreases during heat stress (Cobble and Herman, 1951; Johnson et al., 1966).

Turnover of Digesta Through the Rumen

Development of mathematical models for evaluating the relationship of rate and extent of digestion requires that disappearance of feed from digestive tract be separated into

components and reactions that can be conceptualized and defined (Mertens and Ely, 1982). Models of ruminant digestion should include the major processes that result in disappearance of feed from the digestive tract, which are breakdown and absorption due to digestion and removal due to passage. Digestion in the rumen can be divided into two phases, bacterial breakdown or attachment and chemical breakdown. Passage of undigested residues can be separated into particle size reduction, escape from the rumen and movement of particles through the tract.

Two rate constants can be obtained from the cumulative excretion curve of a marker in the feces of sheep or cattle after a single dose of a marker into the reticulo-rumen (Grovum and Williams, 1973). Two compartmental systems have been used to describe the changes in concentration in fecal dry matter because good fits of the calculated curves (ascending and descending portions) to results were produced. There are various opinions as to what organs the mathematics used to describe recorded events actually refer to.

According to Blaxter et al. (1956), events in the rumen are the predominate factors accounting for variations in rate of passage. The faster turnover rate represents rumen exit, the slower turnover rate represents exit from the abomasum. There is also a time delay for marked particles to travel from the duodenum to the feces.

Hungate (1966) interpreted the faster turnover to represent the rate that large particles were comminuted to particles sufficiently small to pass to the small particle-liquid pool. Slower turnover

rate represents mixing of small particles till they escape via the reticulo-omasal orifice.

Grovum and Williams (1973) introduced and sampled markers from various segments of the gastrointestinal tract. They proposed that the slower turnover compartment represents the rumen, the faster compartment the cecum and proximal colon and the time delay or displacement flow to be the segment connecting the two and the distal large intestine.

Matis noted that processes such as mixing of particles and reduction in size alters the probability of their escape from the rumen and use of a gamma distribution would better represent the "time dependency" of this rate (Ellis et al., 1979). Previous authors used the exponential distributions representing age independent rates to describe the turnover of particles from the rumen. Matis assumed age dependency turnover, the rates that newly ingested particles mix with existing particles and are broken down to smaller size, was associated with the faster turnover compartment. Age independent turnover, the rate that particles exit the rumen, was assumed to be associated with the slower turnover compartment.

Mertens and Ely (1979) have proposed a model of fiber digestion and passage that contains large, medium and small particle compartments within the rumen, followed by a single compartment that represents the large intestine.

Rumen turnover or dilution rate is a measure of the time required for the input of enough of a component to equal that in the rumen (Bull et al., 1979). Usually turnover rate is expressed as

percent per hour. Mean retention time is the inverse of turnover rate. Passage rate is affected by type of marked used, daily intake, physical form of the diet and rumination differences among animals (Mertens and Ely, 1982).

Ruminal contents can be visualized as consisting of liquid and solid phases (Evans, 1981). Liquid phase contains water, soluble feed components and nutrients solubilized by microorganisms. Solid fraction contains undegraded and indigestible material. Liquid material usually leaves the rumen faster due to water intake and salivary secretion. A large part of the bacteria in the rumen are associated with the fluid phase (Cheng et al., 1977; Owens and Isaacson, 1977). The amount of microbial protein synthesized per unit of carbohydrate fermented increases with liquid dilution rate (Isaacson et al., 1975). Efficiency of microbial protein synthesis increases by the decrease of microbial maintenance requirements with increased liquid dilution rate.

The disappearance of particulate digesta involves a number of factors (Evans, 1981). Solid material is solubilized via the degradive actions of microbes. During conversion of feed to bacterial cells, gaseous products of fermentation are formed. Undegraded material, indigestible material and microbes that adhere to the particles go from the rumen into the lower gastrointestinal tract. Only disappearance of undigestible fiber can be used to determine turnover (Ellis et al., 1979). Particle size and specific gravity are two primary factors controlling potential exit of feed

particles from the reticulo-rumen. Material does not leave the rumen until proper particle size is reached (Bergen and Yokoyama, 1977).

Evans (1981a, 1981b) and Warner (1981) summarized correlations from the literature between dietary attributes and measurements of turnover for both liquids and solids in cattle and sheep. The principal relationships of statistical significance were positive correlations between turnover of both liquids and solids and level of dietary intake and proportion of forage in the diet. Negative correlations were observed for the relationship between turnover of both liquids and solids and measure of the digestibility of the diet. Dietary factors such as level of intake (Grovum and Williams, 1977) and the physical component of the diet (Hartnell and Satter, 1979a) have been shown to affect liquid dilution rate. Solid turnover rate is also influenced by level of feed intake (Grovum and Williams, 1977; Owens et al., 1979), physical form of the diet and dietary roughage level. These responses could be interpreted as those of a compartment whose capacity for digesta volume and turnover is regulated by volume constraints.

Liquid dilution rates for cattle averaged 7.5%/h (Evans, 1981a) and mean retention time for solids averaged 25.6 h (Evans, 1981b).

By increasing turnover rates of liquid and solid digesta, the efficiency and quantity of bacterial protein synthesis is increased as is bypass of feed protein (Owens and Isaacson, 1977). Less starch is stored within the bacterial cells, bypass of feed starch increases and the efficiency of digestion and absorption of starch within the small intestine increases as flow from the rumen increases. Fiber

digestion in the rumen decreases as does total tract digestibility as turnover increases, however, intake may be increased.

Several experiments have shown that ruminal volatile fatty acid composition is affected by liquid dilution rate. In general, as liquid dilution rate increases, proportion of acetate increases and proportion of propionate decreases (Harrison et al., 1975; Thomson et al., 1975; Rogers et al., 1979; Rogers and Davis, 1982a, 1982b). The negative effect of dilution rate on percentage of propionate may be due to the fact that succinate, a precursor of propionate, decreases because the increased liquid turnover rate lowers the population of bacteroides and other ruminal bacteria that produce succinate (Thomson et al., 1978). Others have reported an increase in percent propionate with increased liquid dilution rate (Hodgson and Thomson, 1972; Isaacon et al., 1975; Czerkawaski and Breckenridge, 1977; Rogers et al., 1982; Croom et al., 1982).

There is a positive correlation between the molar percentage of ruminal acetate and percent milk fat (Davis, 1979). Manipulation of dilution rate could be important in the prevention of low-milk fat syndrome found in cows consuming high levels of concentrate.

Chalupa (1977) citing data of Harrison et al. (1975) calculated that energetic efficiency of ruminal fermentation was lower at higher dilution rate because less metabolic hydrogen was recovered as VFA. However, more alpha-linked glucose polymer escaped fermentation and total ruminal energy output was similar to lower dilution rate.

There are numerous reports for sheep and cattle relating level of feed intake with turnover rates and mineral salts or salts of

saliva with turnover rates. Van Soest (1975), Hungate (1966), Hogan and Weston (1967), Grovum and Williams (1977), Owens and Isaacson (1977), Chalupa (1977), Bull et al. (1979) and Haaland and Tyrrell (1982) reported positive correlations between feed intake and increased turnover rate of digesta through the rumen.

Potter et al. (1972), Hemsley et al. (1975), Harrison et al. (1975), Thomson et al. (1978), Rogers et al. (1979), Haaland and Tyrrell (1982), Rogers and Davis (1982a, b), and Rogers et al. (1982) reported increased turnover rates with feeding or infusion of NaCl, $NaHCO_3$ or salts of artificial saliva. Hartnell and Satter (1979) did not measure differences in solid turnover rate for higher levels of intake with lactating dairy cows. Bergen (1972) reported no effect of feeding enough NaCl to increase ruminal osmotic pressure to 400 mOsm on liquid dilution rate. In the above reports, at least 3% $NaHCO_3$ was fed. Erdman et al. (1982), Adams et al. (1980) and Stokes et al. (1980) feeding about 1% $NaHCO_3$ did not detect increased liquid dilution rate compared to controls.

Ruminal Buffering Capacity and $NaHCO_3$

Acid-base status and buffering capacity of the rumen is a product of the relationship among amount and rate of saliva secretion, composition and rate of microbial metabolism. This is further compounded by intake and physical characteristics of the diet (Kromann, 1976). Ruminal pH normally ranges from 5.5 to 7.3 (Kay and Hobson, 1963). Ability of the rumen to maintain pH is affected by factors which alter amounts and/or quantity of saliva secretion,

concentration of volatile fatty acids and passage of digesta out of the rumen. Buffering capacity is inversely related to dietary dry matter and energy intake due to effects on saliva secretion and volatile fatty acid concentration in the rumen. Bicarbonate and phosphate ions of saliva are more important in buffering ruminal contents at higher pH while volatile fatty acids have more effect below pH 5.5 (Emmanuel et al., 1969; Van Campen, 1976).

Volatile fatty acid concentration is influenced by energy intake, rate of production and absorption, liquid dilution rate and time postprandial. The pH of the rumen was reduced when finely ground pelleted roughages or high energy feeds were fed (Kromann, 1976). Ruminal pH not only influences microbial fermentation, but volatile fatty acid absorption. As pH decreases, volatile fatty acid absorption increases (Gray, 1948). Rate of absorption of each volatile fatty acid decreases as carbon chain length increases. So the rate of absorption of individual volatile fatty acid influences concentration and therefore buffering capacity. Volatile fatty acid absorption is coupled with CO_2 transfer from blood into the rumen and bicarbonate formation which further influences buffering capacity (Kromann, 1976).

Ruminal pH is the result of interaction among dietary energy intake, physical form, saliva production, microbial metabolism, volatile fatty acid production, pCO_2, volatile fatty acid absorption and bicarbonate exchange across ruminal epithelium. The pH varies with location in the rumen and time postprandial. There is a general inverse relationship between pH and volatile fatty acid concentration

(Emmanuel et al., 1969). Lactic acid accumulates in the rumen of animals fed high energy diets and is usually associated with pH levels less than 5.5. The pH usually reaches a low 2 to 6 hours after feeding depending on the nature of the diet and the rapidity with which a meal is consumed (Briggs et al., 1957). Ruminal pH is lower when animals are fed high concentrate diets compared to high roughage diets (Kromann, 1976; Raun et al., 1962; Luther and Trenkle, 1967).

When high roughage diets are fed, pH of rumen fluid tends to remain relatively high with acetate being the predominate fatty acid (Van Campen, 1976). As more concentrates are fed, propionate and butyrate tend to increase relative to acetate.

In studies where cows were fed high-grain restricted roughage rations, addition of $NaHCO_3$ increased ruminal acetate and decreased propionate (Emery and Brown, 1961; Miller et al., 1965; Esdale and Satter, 1972; Davis, 1979; Erdman et al., 1982a; Snyder et al., 1983).

There has been some controversy over the palatability of bicarbonates. Davis et al. (1964) fed concentrate mixes with 1.5% each of $NaHCO_3$ and $KHCO_3$. Cows would not eat until the buffer levels were halved. Stout et al. (1972), in a study designed to investigate palatability, found that given a choice cows consume more of a concentrate mixture without buffers than ones with 1.5% each of $NaHCO_3$ and $KHCO_3$ or MgO. Erdman et al. (1982b) reported a decreased feed intake with sudden inclusion of 1.5% $NaHCO_3$ in concentrates fed to cows whereas there was no decrease in feed intake to gradual

increases of $NaHCO_3$ in concentrates up to 1.5%. In these studies, concentrates were fed separate from forage. In total mixed rations, palatability would be less of a concern.

Most research involving sodium bicarbonate with dairy cows has focused on prevention of the milk fat depression that results from high concentrate rations. Early lactation is a critical period for lactating cows. She is subject to stress of producing large amounts of milk with a negative energy balance. Frequently there is an abrupt postpartum ration change to a different forage and a ration high in energy, a dietary stress similar to when a beef animal enters a feedlot and is shifted from a high roughage to a high concentrate diet. Research with beef cattle has shown that beneficial effects of $NaHCO_3$ are most pronounced during the first few weeks of feeding period and when animals are abruptly shifted to high energy diets (Emerick, 1976; Huntington et al., 1977; Dunn et al., 1979).

With dairy cows, Erdman et al. (1980) fed alfalfa hay diets prepartum then abruptly switched cows at 4 days postpartum to 40% corn silage and 60% concentrate ration with 0% buffer, 1.5% $NaHCO_3$, 0.8% MgO or 1.5% $NaHCO_3$ and 0.8% MgO. Cows fed $NaHCO_3$ consumed more feed and produced more 4% fat-corrected milk. In a second experiment (Erdman et al., 1982b), feed intake was not increased, but percent milk fat was higher with $NaHCO_3$. Kilmer et al. (1980) abruptly switched cows to high concentrate diets postpartum and found cows fed $NaHCO_3$ increased feed intake faster and had higher milk yields compared to controls. In a second experiment, Kilmer et al. (1981)

fed 50% corn silage and 0.8% $NaHCO_3$ and reported trends towards increased dry matter intake and milk yield.

Emery and Brown (1961) found both $NaHCO_3$ and $KHCO_3$ effective at 454 g/d. Bull et al. (1978) fed mid-lactation cows 60% corn silage and 40% concentrates and reported higher dry matter intake with 2.5% $NaHCO_3$ versus controls or 3.0% limestone.

Donker and Marx (1980) fed 62 cows 50% alfalfa haylage and 50% corn silage plus 1.5% $NaHCO_3$ in the concentrate and reported higher milk yield for the entire lactaion compared to no $NaHCO_3$. However, in a subsequent trial over two complete lactations with 0, 1.5 and 2.5% $NaHCO_3$ in the concentrates there were no differences in feed intake, milk yield or percent milk fat for treatment versus control (Donker and Marx, 1985).

Chase et al. (1981) fed 149 cows 50% corn silage and 0, .4, .8, and 1.6% $NaHCO_3$. With 1.6% $NaHCO_3$, cows had lowest dry matter intake/body weight, but highest fat test. Rogers et al. (1982) fed 108 cows 40% corn silage with 1.2% $NaHCO_3$ in the concentrate. Treatment resulted in higher dry matter intake for the first 7 weeks postpartum and higher milk yield for the first 9 weeks. However, fat-corrected milk and percent milk fat were higher for the whole experiment. Hawkins (1982) fed 50 cows 52% corn silage and 1% $NaHCO_3$ and reported higher milk yield for cows receiving $NaHCO_3$ for the entire lactation. Snyder et al. (1983) reported that 1.2% $NaHCO_3$ with 50 or 75% corn silage increased percent milk fat and 4% fat-corrected milk, but dry matter intake was not affected. Okeke et al. (1983) feeding 60% roughage diet reported no effect on feed intake or

milk yield for 2.5% $NaHCO_3$; however, liquid dilution rate was increased.

A few studies have investigated $NaHCO_3$ during heat stress. Stanley et al. (1972) reported that $NaHCO_3$ effectively offset effects of high concentrate and poor quality forage and raised percent milk fat and 4% fat-corrected milk yield. Harris et al. (1979) added $NaHCO_3$ to rations with various fiber sources with and without the addition of dietary fat for cows past peak milk yield and found no effect of the buffer. Coppock et al. (1982a, 1982b) feeding 1.45% $NaHCO_3$ reported no effect on feed intake, milk yield or percent milk fat. Escobosa et al. (1984) fed 1.7% $NaHCO_3$ and also reported no effect on production responses.

CHAPTER II
INFLUENCE OF DIETARY SODIUM AND POTASSIUM BICARBONATE AND TOTAL POTASSIUM ON HEAT-STRESSED LACTATING DAIRY COWS

Introduction

Summer environment in Florida causes significant decline of milk production. During high environmental temperatures, homeostatic mechanisms strive to maintain homeothermy by reducing feed consumption (Collier et al., 1982) and increasing respiration rate (Bianca and Findlay, 1962). These events do not optimize metabolic functions for milk production.

In one study, voluntary forage intake decreased about 10% for cows not having access to shade compared to those with shade (Roman-Ponce et al., 1977). Milk yield was 6% lower without shade. McDowell (1972) reported a 5% reduction of concentrate consumption and a 22% decline of hay consumption when a lactating Holstein cow was housed in an environmental chamber at 30°C compared to 18°C. With complete mixed diets fed to lactating Holstein and Jersey cows kept in either shade or no shade environments, daily dry matter intake decreased 13% in no shade compared to shade (Beede et al., 1981). Increased respiration rate and water intake that result from increased environmental temperature are surmised also to contribute to reduction of feed intake (Collier et al., 1982; Roman-Ponce et al., 1977).

Panting during heat stress tends to alter alveolar ventilation, subsequently affecting blood pH and CO_2 partial pressure and concentration (McDowell, 1972). Carbon dioxide is eliminated more quickly than normal during accelerated respiration, plasma carbon dioxide partial pressure (pCO_2) is lowered, and blood pH tends to rise. Dale and Brody (1954) found in dairy cattle that ability of blood to take up CO_2 decreased with thermal stress and was associated with a rise in blood pH. Reduced CO_2 combining capacity with higher blood pH was termed respiratory alkalosis. Decreased pCO_2 reduces renal tubular acid secretion exaggerating compensatory loss of alkali reserve in urine (Vander, 1980). We and others (Erdman et al., 1982) hypothesized that resulting increased loss of carbon dioxide may decrease the bicarbonate pool available for buffering in the rumen via salivary secretion. Estimated daily saliva production, 117 to 183 kg in dairy cattle (Bartley, 1976; Meyer et al., 1964), would contain considerable bicarbonate (Bartley, 1976; Hersey et al., 1966), a major buffering agent in the acid-generating ruminal ecosystem. Niles et al. (1980) and Bandaranayaka and Holmes (1976) showed lower ruminal pH in heat-stressed dairy cows. This may be important if energy density of diets for heat-stressed cattle is increased by adding higher proportions of concentrates which may lower ruminal pH as well (Dirkson, 1970). Therefore, dietary supplementation with bicarbonate salts may be warranted. Findlay (1958) reported increased salivary polyrrhea during thermal polypnea in cattle.

This saliva loss may amount to 18 kg/day, equivalent to a daily loss of 50 to 80 g of minerals (Bonsma, 1958).

Current dietary recommendation for potassium for all classes of dairy animals is .8% of dry matter (NRC, 1978). This is based on research with normothermic animals (Erdman et al., 1980; Dennis and Hemken, 1978; NRC, 1978; Dennis et al., 1976). However, potassium is a particularly dynamic cation (Ward, 1966), and at least three factors could contribute to a deficiency in the lactating cow. First, empirical calculation suggests potassium secretion via milk can account for 15 to 40% of total daily potassium intake. Second, high environmental temperatures may increase the animal's daily potassium requirement because of increased potassium loss via sweating (Mallonee, 1984; Singh and Newton, 1978; Jenkinson and Mabon, 1973; Johnson, 1970). Third, feeding feedstuffs that may be low or marginal in potassium represent a potential for some degree of dietary potassium deficiency (Hemken, 1980; Hutcheson, 1980; Linsner, 1980).

Objectives of our experiment were to evaluate effects of heat stress and dietary sodium bicarbonate ($NaHCO_3$), potassium bicarbonate ($KHCO_3$), and total dietary potassium (K) content on production responses, acid-base status, and mineral metabolism of lactating Holstein cows.

Materials and Methods

Experimental design was a split-plot in which environment was no shade (NS; open lot) or shade (S; shade structure with adjoining open

lot) (Roman-Ponce et al., 1977). Within each environment, there was a 2 x 2 x 2 factorial arrangement of dietary treatments. Nine Holstein cows were assigned randomly to each environment for the entire experiment and to a different dietary treatment in each of three 35-day periods from June through September.

Formulation of the basal diet is in Table 2-1. Laboratory analysis of this diet is in Table 2-2. Factorial arrangement of eight (2 x 2 x 2) dietary treatments (Table 2-3) consisted of 0 or .85% added sodium bicarbonate ($NaHCO_3$), 0 or 1.0% added potassium bicarbonate ($KHCO_3$), and 1.0 or 1.5% total dietary potassium (K). Sodium bicarbonate, $KHCO_3$, and KCl were added to the basal diet (diet 1) by replacing ground corn to make other dietary treatments. Formulated additions of $NaHCO_3$ and $KHCO_3$ contributed equal quantities of bicarbonate (.61% of total dry matter). Both KCl and $KHCO_3$ contributed to total dietary K content of diets 5, 6, 7, and 8. All diets were made isochloridic by appropriate additions of NaCl; Na content varied among dietary treatments (.34 to 1.20%).

Ad libitum individual cow daily feed intake and refusals were monitored at 0900 and 1600 h utilizing magnetic gate feeders (American Calan, Inc., Northwood, NH). Feed intake, milk yield, and hourly black globe temperatures (BGT) (readings 0900 to 1700 h) were recorded during the last 2 weeks of each period. The BGT is correlated more highly with responses of cattle to environmental heat

Table 2-1. Formulation of basal diet.

Ingredient	% of dry matter
Cottonseed hulls	25.00
Ground corn	58.82
Corn gluten meal	10.30
Urea	1.00
Limestone	.87
Vitamin A, D, E premix[a]	.99
Dicalcium phosphate	.55
Magnesium oxide	.24
Trace mineral NaCl	.24
Potassium chloride	.79
Sodium chloride	1.20

[a] Provided 4000 IU vitamin A, 2500 IU vitamin D, and 20 IU vitamin E/kg diet dry matter.

Table 2-2. Laboratory analysis of basal diet.

Component	% of dry matter
Crude protein	17.30
Acid detergent fiber	16.20
Calcium	.94
Phosphorus	.41
Magnesium	.23
Potassium	1.00
Sodium	.72
NE_l (Mcal/kg)[a]	1.7

[a] Estimated net energy for lactation.

Table 2-3. Description of dietary treatments as percent of diet dry matter.

Diet	$NaHCO_3$[a]	$KHCO_3$[a]	Total K[a]	KCl[a]	Total Na[b]	NaCl[b]	Na/K
1 (basal)	0	0	1.0	.79	.72	1.20	.72
2	0	0	1.5	1.57	.34	.63	.23
3	.85	0	1.0	.79	.92	1.25	.92
4	.85	0	1.5	1.57	.65	.63	.43
5	0	1.0	1.0	0	.91	1.88	.91
6	0	1.0	1.5	.79	.70	1.25	.47
7	.85	1.0	1.0	0	1.20	1.87	1.20
8	.85	1.0	1.5	.79	1.05	1.30	.70

[a] $NaHCO_3$ and $KHCO_3$ contributed equal quantities of bicarbonate (.61% of total dry matter); KCl and $KHCO_3$ both contributed to total dietary potassium in diets 5, 6, 7, and 8.

[b] All diets were isochloridic by addition of sodium chloride; sodium content varied.

stress than other indices, e.g., dry bulb or temperature humidity index (Buffington et al., 1981). Milk samples (evening milking, 1700 to 1800 h; morning milking, 0500 to 0600 h) were collected the last 3 days of each period for mineral analysis. On the final day of each period between 1200 to 1400 h, rectal temperatures and respiration rates were measured. Blood samples were taken from the caudal vein for plasma mineral analyses. Additional whole blood samples were taken in glass syringes, capped, and iced (4°C) for immediate blood gas analyses. Whole blood pH and gases were analyzed by a semiautomated system (Corning Medical 165/2 blood gas analyzer, Medfield, MA). Blood and milk samples were wet-ash digested in nitric acid, diluted, and analyzed for Na and K by atomic absorption spectrophotometry (Model 5000, Perkin-Elmer, Inc., Norwalk, CT). Data were analyzed by method of least squares analysis of variance (Table 2-4) and general linear model procedures of SAS (SAS, 1982).

Results and Discussion

Environmental Effects

Results of environmental effects on BGT, rectal temperatures, respiration rates, and blood pH and gas compositions are in Table 2-5. Average BGT between 0900 and 1700 h was higher in NS than S. Rectal temperatures were higher for cows in NS than S. Although respiration rates for cows in S were elevated slightly, respiration rates for cows in NS were much higher indicating hyperthermia ($P<.01$).

Table 2-4. Model for least squares analysis of variance.

Source	df
Environment	1
Cow (environment)[a]	15
Period	2
Period x environment	2
Dietary treatment[b]	7
Treatment x environment	7
Residual[c]	17

[a] Error term for environment.

[b] Orthogonal comparisons for dietary treatment effects and interactions.

[c] Error term for all effects except environment.

Table 2-5. Environmental effects on black globe temperature (BGT), rectal temperature, respiration rate, hematocrit, and venous blood pH, pCO_2, HCO_3, and total CO_2 concentrations.

Item	Shade	No shade
Average BGT, °C[a]	29.1	41.0**
Rectal temperature, °C	39.2	40.8**
Respiration rate, per min	83	133**
Hematocrit, %	31.9	32.5
Blood pH	7.43	7.48**
Blood pCO_2, mm Hg	39.8	30.9**
Blood HCO_3, mmol/liter	25.5	22.1**
Blood TCO_2, mmol/liter	26.7	23.1**
HCO_3/pCO_2[b]	21.6	24.3*

* $P<.05$

** $P<.01$

[a] Mean of hourly readings 0900 to 1700 h during final 14 days of each experimental period.

[b] Calculated as described by Davenport (1974).

Blood pH, pCO_2, and HCO_3 concentrations are fundamental to characterization of acid-base status (Davenport, 1974; Kronfeld, 1975). Ideally, arterial blood should be used, but venous blood, which is more readily accessible, is acceptable for some analytical purposes in cattle (Kronfeld, 1975). Blood bicarbonate concentrations for cows in S were in normal range of homeothermic animals not experiencing alkalosis (Phillips, 1970). However, NS cows had higher blood pH, lower pCO_2, lower HCO_3 concentrations, and lower total blood CO_2 concentrations than cows in S. In addition, normal ratio between blood HCO_3 and pCO_2 is about 20:1 (Davenport, 1974). As a result of higher respiration rates, blood pCO_2 decreases more rapidly than HCO_3 content. Ratio of HCO_3 and pCO_2 increases resulting in a physiological condition approaching respiratory alkalosis. In our experiment, a higher HCO_3 to pCO_2 ratio was observed in NS than S (Table 2-5).

Blood pH was higher and pCO_2 lower for cows fed dietary $KHCO_3$ (Table 2-6). An interaction of environment by dietary $NaHCO_3$ was detected for blood bicarbonate ($P<.05$). Without dietary $NaHCO_3$, blood bicarbonate was 18% lower in NS than S; with dietary $NaHCO_3$, cows in NS had blood bicarbonate contents averaging only 2.5% lower than S. No other dietary treatment effects on blood gas measurements were detected.

Supplementation of a bicarbonate buffer to an already potentially alkalotic animal may be of questionable value. However, it appears that during heat stress lactating dairy cows are extremely effective at withstanding dietary challenges to acid-base

Table 2-6. Dietary treatment effects on blood pH, HCO_3, pCO_2, and total CO_2 concentrations.[a]

Dietary treatment[b]	% of Dry matter	pH	HCO_3	pCO_2	Total CO_2	HCO_3/pCO_2
			(mmol/ liter)	(mm Hg)	(mmol/ liter)	
$NaHCO_3$	0	7.46	24.1	34.4	25.2	23.3
	.85	7.46	23.5	35.1	24.6	22.3
$KHCO_3$	0	7.44*	24.7	37.0*	25.8	22.2
	1.0	7.47	23.0	32.5	24.0	23.5
Total K	1.00	7.46	23.4	34.7	24.5	22.4
	1.50	7.46	24.3	34.8	25.3	23.3

* $P<.05$

[a] Least squares means.

[b] Dietary main effects pooled over environments.

homeostasis. Our data indicated no effects of feeding $NaHCO_3$ on blood pH, HCO_3, pCO_2, total CO_2, or ratio of HCO_3/pCO_2 (Table 2-6). Similarly, Erdman et al. (1982) fed 1.0% $NaHCO_3$ to normothermic cows and detected no changes of blood pH, pCO_2, or HCO_3 concentrations. Kellaway et al. (1977) found blood pH increased from 7.37 to 7.39 in calves fed 3% $NaHCO_3$.

Production Responses

Production responses to environment and dietary treatments are in Table 2-7. Mean daytime feed intake of cows in S was 132% higher than NS ($P<.01$). There was no difference in nighttime intake between environments, but total daily intake was 23% higher ($P<.05$) in S. Cows in S produced 19% more milk during evening milking, and 20% more milk during morning milking ($P<.05$) compared to NS. These results were similar to other experiments (Beede et al., 1981; Roman-Ponce et al., 1977; Bandaranayaka and Holmes, 1976; Bayer et al., 1960) where heat-stressed dairy cows had lower production than normothermic cows.

Daytime feed intake was higher ($P<.01$) for cows consuming $NaHCO_3$ than for those not consuming $NaHCO_3$ (Table 2-7). Feed intake at night was not influenced by $NaHCO_3$, but total daily intake was 7.2% higher with $NaHCO_3$ ($P<.05$). Total milk yield and yield at each milking were higher ($P<.05$) for cows receiving dietary $NaHCO_3$. Sodium bicarbonate consumption may have aided ruminal buffering mechanisms during stressful hours resulting in higher daytime feed intake and milk production.

Potassium bicarbonate supplementation (1.0% of diet dry matter) had negative effects on total feed intake and milk yield

Table 2-7. Environmental and dietary treatment main effects on daytime, nighttime and total daily feed intake and milk yield.

Environment		Feed intake			Milk yield[b]		
		Day[c]	Night[c]	Total	Evening[d]	Morning[d]	Total
Shade, 29.1°C[e]		7.9**	13.6	20.7*	9.2*	10.7*	19.4*
No shade, 41.0°C[e]		3.4	13.6	16.8	7.7	8.9	17.0
Dietary treatment[f]	% of dry matter						
$NaHCO_3$	0	5.2**	13.4	18.1*	8.3*	9.6*	17.9**
	.85	6.2	13.8	19.4	8.6	10.1	18.5
$KHCO_3$	0	5.6	14.0	19.4**	8.6*	10.1*	18.6**
	1.0	5.8	13.2	18.1	8.2	9.6	17.8
Total K	1.0	5.6	13.9	19.1	8.4	9.7*	18.0*
	1.5	5.7	13.3	18.5	8.5	10.0	18.5

a Least squares means.

b Feed intake included as covariate.

c Day (0900 to 1600 h); night (1600 to 0900 h).

d Evening milking (1700 to 1800 h); morning milking (0500 to 0600 h).

e Mean hourly black globe readings taken between 0900 to 1700 h the last 14 days of each experimental period.

f Dietary main effects pooled over environments.

* Signifies difference between pairs of main effect means ($P<.05$).

** Signifies difference between pairs of main effect means ($P<.01$).

Table 2-7). Total daily feed intake was lower ($P<.01$) as was daytime and nighttime milk production ($P<.05$) for cows consuming $KHCO_3$. Lower palatability of $KHCO_3$-containing diets may explain decreases of feed intake for cows offered treatments 5, 6, 7, and 8 (Table 2-3). However, Emery and Brown (1961) did not report lower feed intake when $KHCO_3$ was added to diets based on alfalfa hay or pellets; likewise, Downer et al. (1983) reported no effects of 1.5% $KHCO_3$ on milk fat content when $KHCO_3$ was fed in a potentially fat-depressing diet containing silage. Our basal diet contained cottonseed hulls as roughage, which may not have masked potential unpalatability of $KHCO_3$. There was interaction of $KHCO_3$ by total dietary K on total daily feed intake ($P<.05$). Feed intake decreased 14% (19.8 vs. 17.0 kg/day) with 1.5% total dietary K when $KHCO_3$ supplied additional K; if KCl supplied supplemental K at either 1.0 or 1.5% total dietary K, feed intake was not affected. This may explain change of the trend compared to the study of Beede et al. (1981) where cows consumed more feed (3.7, 4.0, and 4.7 kg) during heat stress with .66, 1.08, and 1.64% total dietary K, with KCl as the supplemental K source. Despite a trend for lower feed intake for cows consuming 1.5% total dietary K, morning and total daily milk production were greater ($P<.05$). Both total dietary K concentrations in our experiment were greater than the current recommendation (NRC, 1978).

Mineral Metabolism

Plasma Na concentrations for cows in NS were about 5% lower than in S ($P<.05$, Table 2-8). Hyperthermic cows apparently excrete lower concentrations and absolute quantities of K in urine than

Table 2-8. Environmental and dietary treatment main effects on plasma and milk sodium and potassium.[a]

Environment		Plasma		Milk	
		Na	K	Na	K
		---------- (ppm) ----------			
Shade (29.1°C)[b]		3140*	232	512*	1877
No shade (41.0°C)[b]		2992	229	438	1895
Dietary treatment[c]	% of dry matter				
$NaHCO_3$	0	3010	231	479	1867
	.85	3122	231	471	1905
$KHCO_3$	0	3073	230	471	1895
	1.0	3059	232	479	1877
Total K	1.0	3112	231	473	1844*
	1.5	3020	231	477	1928

* $P<.05$.

[a] Least squares means.

[b] Mean hourly black globe readings taken between 0900 to 1700 h during final 14 days of each experimental period.

[c] Dietary main effects pooled over environments.

normothermic cows (El-Nouty et al., 1980). Also, during chronic heat stress, plasma concentrations of aldosterone decreased (El-Nouty et al., 1980; Israel et al., 1978). As plasma aldosterone concentrations increased, urinary Na decreased and K increased (Edelman and Marver, 1980; Guyton, 1976). Other reports suggested otherwise with regard to renal K excretion (Rabinowitz and Gunther, 1978; MacFarlaine, 1968; Lippsett et al., 1961). Apparently, Na and K (in relation to hydrogen ions) are excreted or reabsorbed reciprocally at the distal tubule of the kidney (Masero and Siegel, 1971). This may serve as a survival mechanism to conserve K for other higher-priority physiological demands such as neural and muscular functions and possibly loss in sweat during body cooling. Conservation of K during heat stress suggests increased renal requirement for Na due to the reciprocal Na excretion and K reabsorption mechanism. Lower plasma Na for cows in NS suggested increased Na excretion by the kidney. Additionally, plasma volume may be increased during heat stress (El-Nouty et al., 1980), thus diluting the absolute quantity of plasma Na. However, in our experiment a similar effect was not observed for plasma K, and there was no difference in hematocrit due to environment (Table 2-5). There were no dietary effects on plasma Na or K concentrations.

Sodium concentrations in milk from cows in NS were 14% lower than from cows in S ($P<.05$, Table 2-8). This was similar to environmental effects on plasma Na. There were no effects of dietary treatment on milk Na concentrations. Much higher than recommended (NRC, 1978) dietary Na (.34 to 1.20% in our experiment) had no effect

on milk Na content; concentrations in normal range (450 to 580 ppm) were reported by others (Larson and Smith, 1974; Triebold and Aurand, 1963). There were no differences in milk K content due to environment. However, 1.5% total dietary K increased milk K concentrations compared to 1.0% K ($P<.05$).

Summary

Measurements of rectal temperature, respiration rate, and blood gases indicated cows in NS were hyperthermic during part of the day and approached respiratory alkalosis. Plasma and milk Na concentrations were lower for cows in NS than S. This may reflect a renal homeostatic mechanism for conserving K during heat stress at the expense of Na. Dietary treatments had little effect on blood gas measurements.

Feed intake during hours of heat stress was much higher in S than NS. During cooler nighttime hours feed intake was not different between environments, but total daily intake was greater in S than NS. Total daily, evening, and morning milk yields were higher for S than NS.

Supplementation of $NaHCO_3$ (.85% of diet DM) increased feed intake and milk production during day and night hours. Sodium bicarbonate did not affect plasma or milk Na or K concentrations. Potassium bicarbonate supplementation did not affect significantly day or night feed intake but did affect total daily intake of dry matter, and had a negative influence on evening and morning milk production. Potassium bicarbonate did not affect plasma or milk

concentrations of Na or K. Higher percent of total dietary K did not affect feed intake but increased morning and total daily milk yield. This differed from previous research at this experiment station (Mallonee et al., 1985) where KCl was the sole source of supplemental K. In the current experiment, $KHCO_3$ supplied supplemental K in half the diets. Based on results of the current experiment, $KHCO_3$ would not be recommended as a dietary source of K or HCO_3. Total dietary K concentration did not influence plasma Na or K concentrations but increased milk K concentrations. Further research into effects of bicarbonate salts and different dietary minerals on ruminal and body physiology of heat-stressed dairy cows is warranted for a basic understanding of the condition and to determine if potential corrective measures are to be applied.

CHAPTER III
RESPONSES OF LACTATING COWS TO DIETARY SODIUM SOURCE AND QUANTITY AND POTASSIUM QUANTITY DURING HEAT STRESS

Introduction

Sodium (Na) deprivation results in reduced milk yield (Mallonee et al., 1982b; Smith and Aines, 1959). Cows fed a Na-deficient diet for 2 years produced about 50% less milk each year compared to pretrial production with a Na-sufficient diet (Smith and Aines, 1959). Sodium requirements and production responses may be affected by temperature, humidity and sweating rate, by Na concentration in grain, forage and water supply and by stages of lactation, gestation, growth and level of production (Cunha, 1983). Milk contains about .06% Na (Miller, 1979).

Potassium (K) content of milk is about .15% (Miller, 1979) which may represent 15 to 40% of total daily K intake of lactating cows depending on levels of production and feed intake. Only small amounts of K are stored in the body, so quantity of K in the diet can influence amounts of milk produced (International Minerals and Chemical Corp., 1981). Corn silage, cereal grains, and some by-product feeds are low in K and may potentiate a deficiency (International Minerals and Chemical Corp., 1981).

Homeostatic mechanisms attempt to maintain body temperature during heat stress by reducing feed consumption (McDowell, 1972);

consequently intake of essential nutrients is reduced. Furthermore, greater amounts of K are lost through sweating during high temperatures (Mallonee et al., 1985; Jenkinson and Mabon, 1973; Johnson, 1970). Potassium and Na may be important especially during heat stress as they are major regulators of body water balance (Vander, 1980). Sodium is required at the kidney for K conservation and to balance bicarbonate excretion electrically (Vander, 1980; Masero and Siegel, 1977). Potential deficiencies of Na and K may be implicated in lower milk production during hyperthermia.

High producing dairy cows are challenged nutritionally to achieve their genetic potential for milk production by feeding diets high in digestible energy and concentrates. Feeding such diets to heat-stressed cows may be advantageous since heat increment is lower than with higher forage diets, but such diets tend to increase acidic conditions in the rumen. Furthermore, thermally-induced hyperventilation may alter acid-base homeostasis by reducing CO_2 available as a substrate for salivary bicarbonate (Schneider et al., 1984) thus reducing buffering capacity of the rumen. Lower ruminal pH was reported for heat-stressed cows (Niles et al., 1980; Bandaranayaka and Holmes, 1976) and use of dietary buffers may be warranted to overcome this.

Objectives of this experiment were to evaluate effects of source and quantity of dietary Na (sodium bicarbonate and sodium chloride) and total dietary K quantity on acid-base status, production responses, and mineral metabolism of lactating Holstein cows during heat stress.

Materials and Methods

Experimental design was a split-plot in which environments were no shade (NS; open lot) or shade (S; shade structure with adjoining open lot)(Roman-Ponce et al., 1977). Cross-classified with each environment was a 2 x 2 x 2 factorial arrangement of dietary treatments. Twelve Holstein cows were assigned randomly to each environment for the entire experiment and to a different dietary treatment in each of three, 35-day periods from June through September.

Basal diet (Table 3-1) contained approximately 38% corn silage and 62% concentrates, dry basis. This diet met recommended (NRC, 1978) content of Na (Table 3-2) and had greater than recommended K content (NRC, 1978). Factorial arrangement of eight dietary treatments (Table 3-3) consisted of 0 or 1.0% sodium bicarbonate ($NaHCO_3$) added to basal diet, 0 or .73% sodium chloride (NaCl) added to basal diet and 1.3 (basal) or 1.8% total dietary potassium (K). Sodium bicarbonate, NaCl and KCl were added to the basal diet (diet 1) by replacing ground corn. Formulated additions of Na yielded three total dietary Na concentrations, .18 (basal), .55 and .88% of dry matter (average analytical values). Diets were made isochloridic and isonitrogenous by appropriate additions of ammonium chloride and/or urea.

Ad libitum feed intake and refusals were monitored daily for individual cows utilizing electronic gate feeders (American Calan, Inc., Northwood, NH). In S feed bunks were under shade, whereas bunks in NS were covered with clear plastic so as not to cast a shadow. Feed intake, milk yield and black globe temperature (BGT)

Table 3-1. Formulation of basal diet.

Ingredient	% of dry matter
Corn silage	37.9
Ground corn	42.9
Soybean meal	16.5
Limestone	.85
Dicalcium phosphate	.30
Vitamin A, D, E premix[a]	.90
Magnesium oxide	.21
Trace mineral sodium chloride	.25
Potassium chloride	.19
Ammonium chloride	1.50

[a] Provided 3636 IU vitamin A, 2273 IU vitamin E and 18 IU Vitamin D per kg diet dry matter.

Table 3-2. Laboratory analysis of basal diet.

Component	% of dry matter
Crude protein	18.54
Acid detergent fiber	15.30
Calcium	.97
Phosphorus	.37
Magnesium	.29
Potassium	1.30
Sodium	.18
NE_l (Mcal/kg)[a]	1.69

[a] Estimated from formulation.

Table 3-3. Description of 2 x 2 x 2 factorial arrangement of dietary treatments as percent of dietary dry matter.

Diet	Added to basal diet				Total dietary[b]		
	$NaHCO_3$[a]	NaCl[a]	KCl	NH_4Cl	Na	K	Urea
1 (basal)	0	0	.19	1.50	.18	1.3	0
2	0	0	1.16	.78	.18	1.8	.38
3	1.0	0	.19	1.50	.55	1.3	0
4	1.0	0	1.16	.78	.55	1.8	.38
5	0	.73	.19	.83	.55	1.3	.38
6	0	.73	1.16	.14	.55	1.8	.83
7	1.0	.73	.19	.83	.88	1.3	.38
8	1.0	.73	1.16	.14	.88	1.8	.83

[a]Equal Na supplied from either Na source within each total Na level. All diets were isochloridic and isonitrogenous by adding ammonium chloride and/or urea.

[b]Averages of analytical values from subsamples taken throughout the experiment.

readings (1300 to 1500 h) were recorded the last 2 weeks of each period. Milk samples were collected the last 3 days of each period for fat, protein and mineral analyses. On the last day of each period between 1300 and 1500 h, rectal temperatures and respiration rates were measured. Blood samples were taken from the caudal vein for plasma mineral analyses. Additional blood samples were taken in glass syringes, capped, iced and analyzed immediately for pH and gases by a semiautomated system (Corning Medical 165/2 Blood Gas Analyzer, Medfield, MA). Blood and milk samples were wet-ash digested in nitric acid, diluted and analyzed for Na and K by atomic absorption spectrophotometry (Model 5000, Perkin-Elmer, Inc., Norwalk, CT). Data were analyzed by method of least squares analysis of variance (Tables 3-4, 3-5 and 3-9) using general linear model procedures of SAS (SAS, 1982).

Results and Discussion

Acid-Base Status

Measurement of environmental effects indicated greater heat stress for animals in NS than S (Table 3-6). Black globe temperatures in NS between 1300 and 1500 h averaged 40.9°C compared to 30.1°C in S. Shade environment was above the upper critical temperature for lactating cows (Fuquay, 1981) indicating some degree of heat stress. Rectal temperatures and respiration rates were elevated in NS, hematocrit and plasma protein contents were not affected by environment. Dale and Brody (1954) described decreased CO_2 combining capacity of blood with increased blood pH observed during thermal

Table 3-4. Least squares analyses of variance of sources of variation of factors affecting blood gases and blood, urine and fecal pH.

Source	df	Mean squares						
		Blood pH	Blood HCO_3, mmol/liter	Blood pCO_2, mm Hg	Blood TCO_2, mmol/liter	Blood HCO_3/pCO_2	Urine pH[c]	Fecal pH
Environment (E)	1	.0396**	30.337+	502.76**	40.83*	80.58**	.882	.00065
Cow (Environment)[a]	22	.0028	7.778	37.31	8.24	8.50	.361	.00026
Period	2	.0290**	344.074**	555.19**	359.73**	68.62**	2.811**	.00199**
Period x Environment	2	.0005	12.617	27.62	14.70	4.59	.043	.00012
Treatment								
NaCl	1	.0005	42.915*	50.30	49.23*	1.88	.417	.00046+
$NaHCO_3$	1	.0052	69.158**	71.17	65.70**	3.11	3.873**	<.00001
Dietary K	1	.0094*	8.175	1.91	9.42	37.29*	1.020+	.00026
NaCl x $NaHCO_3$	1	.0029	21.794+	15.39	21.26	5.63	.367	.00018
NaCl x K	1	.0001	42.748*	107.00+	43.59*	.27	.115	.00019
$NaHCO_3$ x K	1	.0038	2.596	1.55	3.02	15.68	.076	<.00001
NaCl x $NaHCO_3$ x K	1	<.0001	1.185	16.92	.80	1.82	.874	.00044+
Environment x treatment								
E x NaCl	1	.0070+	.865	32.71	1.21	27.32	.058	.00076*
E x $NaHCO_3$	1	.0034	.026	9.43	.50	3.37	.229	.00002
E x Dietary K	1	.0067+	<.001	20.82	.14	19.44	.967	<.00007
E x NaCl x $NaHCO_3$	1	.0020	.615	7.65	.33	4.22	.750	<.00001
E x NaCl x K	1	.0019	5.792	37.86	8.24	3.53	.183	.00017
E x $NaHCO_3$ x K	1	<.0001	17.802	83.76	18.90	.03	.020	<.00001
E x NaCl x $NaHCO_3$ x K	1	<.0001	16.009	60.33	14.73	1.76	.230	.00017
Residual[b]	29	.0022	7.080	29.37	7.66	7.08	.343	.00012

+ P<.1

* P<.05

** P<.01

[a] Error term for environment.

[b] Error term for all sources of variation except environment.

[c] Degrees of freedom in residual equal 25.

Table 3-5. Least squares analyses of variance of sources of variation of factors affecting daily dry matter intake, milk yield and fat and protein yields and contents.

Source	df	Mean squares								
			Milk yield, kg							
		Dry matter intake,kg	Actual[d]	Actual[d,e]	4% FCM[f]	4% FCM[g]	Milk fat,%[h]	Milk fat yield,kg[h]	Milk protein,%[h]	Milk protein yield,kg[h]
Environment (E)	1	29.66*	3.57	1.41	12.95	.02	.644	.039	.346+	.026
Cow (Environment)[a]	22	5.85*	14.79**	13.29**	10.26**	8.19**	.633**	.023**	.105**	.015**
Period	2	18.40**	63.52**	34.46**	9.84	6.25*	2.430**	<.001	.138**	.058**
Period x Environment	2	2.38	1.76	.17	3.94	.73	.102	.010	.001	.002
Treatment										
NaCl	1	2.06	4.24	8.50*	5.62	10.90*	.001	.010	.034+	.003
$NaHCO_3$	1	3.86	16.71*	7.76*	30.31**	8.40*	.264+	.066**	.011	.021*
Dietary K	1	10.80*	10.05*	1.08	9.19	.05	.002	.014	.133**	.002
NaCl x $NaHCO_3$	1	10.33*	14.02*	2.64	20.12*	1.43	.134	.039*	.004	.021**
NaCl x K	1	.30	2.90	1.85	6.24	4.03	.242+	.015	.012	.005
$NaHCO_3$ x K	1	2.97	2.98	.40	<.01	3.02	.348	.002	.004	.003
NaCl x $NaHCO_3$ x K	1	6.53	2.35	<.01	1.69	2.16	.044	.002	.001	.006
Environment x Treatment										
E x NaCl	1	.19	.53	.99	<.01	.56	.019	<.001	.005	<.001
E x $NaHCO_3$	1	2.94	.24	.32	.05	3.69	.013	<.001	.002	.001
E x Dietary K	1	2.26	8.57+	3.83	18.68*	6.65	.158	.044*	.006	.007+
E x NaCl x $NaHCO_3$	1	.09	.12	.28	.50	.50	<.001	.001	.026	.003
E x NaCl x K	1	.36	1.97	1.05	1.42	.43	.002	.002	.026	.005
E x $NaHCO_3$ x K	1	.54	3.83	2.22	4.89	1.47	.012	.009	.009	.004
E x NaCl x $NaHCO_3$ x K	1	2.93	.63	.07	7.34	.31	.412*	.024	.002	.002
Feed intake[c]	-	-	-	28.62**	-	52.41**	-	-	-	-
Residual[b]	29	2.52	2.32	1.38	3.56	1.75	.069	.008	.011	.003

+ P<.1

* P<.05

** P<.01

[a] Error term for environment.

[b] Error term for all sources of variation except environment.

[c] Continuous independent variable (covariate) for actual milk yield and 4% fat-corrected milk yield (4% FCM).

[d] Actual milk yield, unadjusted for fat content.

[e] Degrees of freedom in residual equal 28.

[f] Milk yield adjusted to 4% fat content; degrees of freedom in residual equal 28.

[g] 4% FCM yield; degrees of freedom in residual equal 27.

[h] Degrees of freedom in residual equal 27.

Table 3-6. Environmental effects on least squares means of black globe temperature (BGT), rectal temperature, respiration rate, hematocrit, blood pH and gases and urinary and fecal pH in S or NS environments.

Item	Shade	No Shade
Average BGT, °C[a]	30.1	40.9**
Rectal temperature, °C	39.8	40.6**
Respiration rate, /min	97	125**
Hematocrit, %	32.6	32.4
Blood pH	7.394	7.443**
Blood pCO_2, mm Hg	36.3	30.5**
Blood HCO_3, mmol/liter	21.8	20.2$^+$
Blood total CO_2, mmol/liter	23.0	21.2*
HCO_3/pCO_2[b]	19.95	22.00**
Urine pH	6.90	7.16
Fecal pH	6.08	5.92$^+$

$^+$ P<.10

* P<.05

**P<.01

[a] Mean of readings at 1300 to 1500 h during final 14 days of each experimental period.

[b] Calculated as described by Davenport (1974).

stress as respiratory alkalosis. In our experiment, blood pH was higher in NS than S cows and pCO_2 was lower, thus indicating a trend toward respiratory alkalosis (Table 3-6). In addition, normal ratio of blood HCO_3 to pCO_2 is about 20:1 (Davenport, 1974). A greater ratio suggests a physiological state approaching respiratory alkalosis. In our experiment blood HCO_3 to pCO_2 ratio was higher in NS than S (Tables 3-4 and 3-6).

Blood HCO_3 and total CO_2 concentrations were higher in cows supplemented with NaCl and $NaHCO_3$ (Tables 3-4 and 3-7). Higher dietary K increased blood pH and HCO_3 to pCO_2 ratio. This effect may have been due to the alkalogenic effects of these cations (Leach, 1979). Contrary to our findings, Escobosa et al. (1984) detected lower arterial pCO_2 and HCO_3 for cows fed diets containing .55 compared to .18% Na. Erdman et al. (1982) reported no change in acid-base status measured by venous blood of cows fed a 1.0% $NaHCO_3$ diet. Total CO_2 of blood is the sum of HCO_3 and dissolved CO_2 (Davenport, 1974). Interaction of NaCl with dietary K was detected for blood total CO_2. Response to .73% NaCl (versus 0%) was greater with 1.3% than with 1.8% dietary K (23.68 vs. 22.73 mmol/liter). At 0% added NaCl total CO_2 was 19.71 mmol/liter at 1.3% K and 22.26 mmol/liter at 1.8% K. This suggests that up to certain concentrations, dietary cations may be associated with an increased blood CO_2. Renal mechanisms might buffer against further alkalogenic effects of higher concentrations of these cations by excretion in urine (Vander, 1980). Urine pH was higher for cows fed $NaHCO_3$, and tended to be higher with 1.8% dietary K and added NaCl (Table 3-7).

Table 3-7. Dietary treatment effects on blood gases, and on urine and fecal pH.[a]

Dietary treatment[b]	% of diet dry matter	pH	HCO_3 (mmol/liter)	pCO_2 (mm Hg)	Total CO_2 (mmol/liter)	HCO_3/pCO_2[c]	Urine pH	Fecal pH
$NaHCO_3$	0	7.408	19.8**	32.2	20.9**	21.0	6.74**	6.01
	1.0	7.429	22.2	34.6	23.3	21.5	7.32	5.98
NaCl	0	7.415	20.0*	32.3	21.0*	21.0	6.92	5.99
	.73	7.422	22.0	34.5	23.2	21.4	7.14	6.00
Dietary K	1.3	7.406	20.3	33.6	21.7	20.4*	6.89+	5.94
	1.8	7.431*	21.4	33.2	22.5	22.0	7.17	6.05

+ $P<.10$

* $P<.05$

** $P<.01$

[a] Least squares means.

[b] Dietary main effects pooled over environments.

[c] Calculated as described by Davenport (1974).

Production Responses

Daily feed dry matter intake was not affected with additions of $NaHCO_3$ or NaCl (Tables 3-5 and 3-8). Cows receiving 1.8% total dietary K consumed 4.5% more dry matter than cows receiving 1.3% K ($P<.05$). Interaction of NaCl with $NaHCO_3$ ($P<.05$) showed that feed intake increased 8.5% (19.2 vs 17.7 kg/d), when 1.0% compared with 0% added $NaHCO_3$ was fed without added NaCl. However, with .73% NaCl, consumption decreased 2.7% (17.7 vs 18.2 kg/d) with 1.0% compared to 0% added $NaHCO_3$. Feed dry matter consumption was similar when neither NaCl or $NaHCO_3$ were added to the basal diet (.18% total Na, Figure 3-1) compared with feeding both salts together (.88%) total Na, Figure 3-1). However, intake was 5.6% higher with .55% Na from either source than with .18% or .88% Na. This indicated .18% total dietary Na limited maximum feed intake, but there was no benefit to .88% compared, with .55% Na. In agreement with previous reports (Mallonee et al., 1985; Schneider et al., 1984; Roman-Ponce et al., 1977) feed consumption was reduced for NS compared to S cows (Table 3-8). However, no environmental by dietary treatment interactions on dry matter intake were detected ($P>.1$, Table 3-5).

Milk yield responses to dietary and environmental treatments were evaluated statistically without and with daily dry matter feed intake as a continuous independent variable (covariate) in the mathematical model (Table 3-5). Actual milk yield (not adjusted to 4% fat content) increased 6.3% with $NaHCO_3$ addition and 4.2% with higher dietary K when feed intake was not included in the model. Effect of NaCl was not detected. However, when feed intake was in

Table 3-8. Dietary and environmental main effects on daily feed dry matter and milk yield and composition.[a]

	Dietary main effects[b]						Environmental effect	
	$NaHCO_3$, %		NaCl, %		Dietary K, %		Shade (30°C)[c]	No shade (41°C)[c]
	0	1.0	0	.73	1.3	1.8		
Feed dry matter intake, kg	17.9	18.5	18.4	18.0	17.8	18.6*	18.8	17.6*
Actual milk yield, kg[d]	18.9	20.1*	19.2	19.8	19.1	19.9*	19.7	19.4
Actual milk yield, kg[e]	19.1	19.9*	19.0	20.0*	19.3	19.6	19.3	19.8
4% FCM, kg[f]	17.1	18.7**	17.5	18.2	17.5	18.3	18.2	17.5
4% FCM, kg[e]	17.5	18.3*	17.3	18.4*	17.9	17.8	18.0	17.7
Milk fat, %	3.42	3.56+	3.49	3.50	3.50	3.48	3.59	3.39
Milk fat yield, kg	.63	.71**	.65	.69	.66	.69	.69	.65
Milk protein, %	3.57	3.54	3.58	3.52+	3.6	3.50**	3.63	3.48+
Milk protein yield, kg	.67	.71*	.68	.70	.68	.69	.71	.67

[a] Least squares means.

[b] Dietary main effects pooled over environments.

[c] Black globe temperatures 1300 to 1500 h the final 14 days of each experimental period.

[d] Actual milk yield not adjusted to 4% fat content.

[e] Least squares means with feed dry matter intake as continuous independent variable (covariate) in analysis of variance.

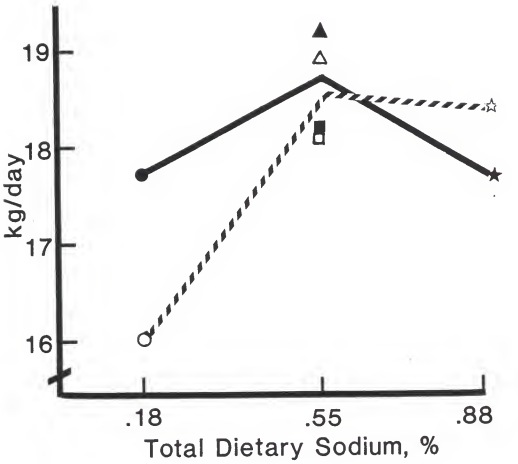
[f] Four percent fat-corrected-milk yield.

+ Signifies difference between pairs of means ($.05<P<.10$).

* Signifies difference between pairs of means ($P<.05$).

** Signifies difference between pairs of means ($P<.01$).

Figure 3-1. Effect of total dietary Na concentration on feed dry matter intake (closed symbols, ——) and 4% fat-corrected-milk yield (open symbols, ----); ●, ○ = basal diet, .18% total sodium; ▲, △ = 1.0% added sodium bicarbonate, .55% total sodium; ■, □ = .73% added sodium chloride, .55% total sodium; ★, ☆ = 1.0% added sodium bicarbonate plus .73% added sodium chloride, .88% total sodium. At .55% total sodium, connecting point of line for each response is the average of responses observed when each sodium salt was fed separately.


19
18
17
16
kg/day
.18
.55
.88
Total Dietary Sodium, %

the mathematical model actual milk yield increased with NaCl and $NaHCO_3$ additions, but not with higher dietary K. This suggested that increase in actual milk yield with higher K was through increased (4.5%) feed intake, whereas responses to added NaCl and $NaHCO_3$ were apparently associated with other factors. Four percent fat-corrected milk yield (unadjusted for feed intake) increased 9.4% with added $NaHCO_3$ but was not affected by added NaCl or dietary K content. With feed intake in the mathematical model 4% FCM yield increased 4.6% and 6.4% with added $NaHCO_3$ and NaCl, respectively; dietary K had no detectable effect. Yield responses to $NaHCO_3$ (Snyder et al., 1983; Erdman et al., 1980) and supplemental K (Chapter II) above current recommendations (NRC, 1978) were reported previously. An interaction of NaCl with $NaHCO_3$ on 4% FCM yield (unadjusted for feed intake) showed an 18.1% increase (18.9 vs 16.0 kg/d) when 1.0% compared to 0% added $NaHCO_3$ was fed without added NaCl (Table 3-5). With .73% added NaCl, 4% FCM yield increased only slightly (1.7%) with 1.0% compared to 0% $NaHCO_3$. As with feed intake, 4% FCM yield was greater with .55% total dietary Na (from either Na source) compared to .18% (Figure 3-1). Fat-corrected milk yield was similar between .55 and .88%. Suggested Na content for lactation diets is currently .18% (NRC, 1978).

In agreement with previous reports (Mallonee et al., 1985; Roman-Ponce et al., 1977; Chapter II) feed intake was lower for NS than S cows (Table 3-8). Effects of environment on milk yield in this experiment were not statistically significant without or with adjustment to equalize feed intake between NS and S (Tables 3-5 and

3-8). Mallonee et al. (1985) also found no difference in milk yield for cows in NS and S environments after adjustment for feed intake. Other reports (Collier et al., 1981; Roman-Ponce et al., 1977; McDowell, 1972; Chapter II) indicated that heat stress reduced milk production. Four percent fat-corrected milk yield responses to higher K differed between environments (environment by dietary K interation, $P<.05$). Four percent fat corrected milk yield (unadjusted for intake) in S increased 9.7% (19.2 vs 17.5 kg/day) with 1.8% compared to 1.3% dietary K, whereas no effect of K was noted in NS (17.4 vs 17.7 kg/day).

Milk fat percent of cows receiving 1.0% $NaHCO_3$ was higher ($P<.06$) which is in accord with earlier work (Escobosa et al., 1984; Erdman et al., 1982; Rogers et al., 1982; Kilmer et al., 1981). There was an interaction of $NaHCO_3$ with dietary K ($P<.05$). Milk fat percent was greater with 1.8% K and 1.0% $NaHCO_3$ (3.64%) compared to 1.8% K and no added $NaHCO_3$ (3.33%). With 1.3% K, however, there was little effect of added $NaHCO_3$. Insofar as a NaCl by dietary K interaction was not detected ($P>.07$), interaction of $NaHCO_3$ with dietary K was likely due to ruminal buffering action of bicarbonate as opposed to effects of the dietary Na cation per se. Environment did not affect milk fat percent (Tables 3-5 and 3-8). Collier et al. (1981) also found no effect of hot environment; however, in other studies heat stress reduced milk fat percent (McDowell, 1972). Milk protein percent decreased with 1.8% dietary K but milk protein yield was not affected by dietary K concentration. Sodium bicarbonate increased milk protein yield 6%, but milk protein percent was

unaffected. Escobosa et al. (1984) found that milk protein percent increased with 1.7% $NaHCO_3$. Rogers et al. (1982) reported that cows consuming 2% $NaHCO_3$ produced 6% more total milk protein than cows fed a basal diet containing no supplemental $NaHCO_3$; however, Fronk et al. (1981) reported no effect. In our experiment milk protein percent was slightly lower in NS than S. Collier et al. (1981) did not detect effects of S or NS on total protein, freezing point depression or somatic cell content of milk.

Mineral Metabolism

Sodium bicarbonate in the diet caused a slight depression in plasma Na concentrations compared to no $NaHCO_3$ (Table 3-9 and 3-10) but concentrations of K were not affected. Escobosa et al. (1984) showed higher concentrations of K in blood plasma of cows fed 1.45% $NaHCO_3$ during natural heat stress and Hemsley et al. (1975), feeding NaCl at 150 g/day to sheep, found increased plasma K but no change in plasma Na. Erdman et al. (1982) observed no effects of 1.0% $NaHCO_3$ on plasma K or Na. Serum cation concentrations and milk production were not affected by feeding diets containing .45, .55 or .66% K (Dennis et al., 1976).

Plasma Na concentrations were lower in NS than S (Tables 3-9 and 3-10) but plasma K concentrations did not differ. This agreed with previous research (Chapter II). With involvement of hydrogen ion, Na and K may be excreted in a reciprocal fashion by the kidney (Masero and Siegel, 1977). Slightly lower plasma Na in NS than S suggested increased Na excretion by the kidney to conserve K for physiological regulation of heat stress-related needs such as sweating

Table 3-9. Least squares analyses of variance of sources of variation of factors affecting plasma and milk mineral concentrations.

Source	df	Plasma Na, ppm	Plasma K, ppm	Milk Na, ppm	Milk K, ppm
Environment (E)	1	143000*	1141.7	8842	395794
Cow (Environment)[a]	22	33655	671.0**	38900**	24443*
Period	2	320208	1739.8**	28922**	289452**
Period x Environment	2	14398	487.1^{+}	2899	12124
Treatment					
NaCl	1	2218	521.9^{+}	4252	687
$NaHCO_3$	1	88414^{+}	443.5	15088*	386
Dietary K	1	12902	75.6	4933	13060
NaCl x $NaHCO_3$	1	25315	70.2	8724^{+}	377
NaCl x K	1	38	14.2	5186	17305
$NaHCO_3$ x K	1	43059	41.4	1219	8704
NaCl x $NaHCO_3$ x K	1	212987*	1048.0*	234	46411*
Environment x treatment					
E x NaCl	1	14534	17.9	37	6445
E x $NaHCO_3$	1	3867	116.6	89	1215
E x Dietary K	1	56992	881.4*	627	9721
E x NaCl x $NaHCO_3$	1	1080	1437.7**	311	183
E x NaCl x K	1	130	1877.7**	7487	351
E x $NaHCO_3$ x K	1	36545	141.6	6820	6341
E x NaCl x $NaHCO_3$ x K	1	4240	15.0	1443	1125
Residual[b]	29	24157	174.5	2649	9825

$^{+}$ P<.1

* P<.05

** P<.01

[a] Error term for environment.

[b] Error term for all sources of variation except environment.

Table 3-10. Environmental and dietary main effects on plasma and milk sodium and potassium concentrations (ppm).[a]

	Dietary main effects[b]						Environmental effects	
	$NaHCO_3$,%		NaCl,%		Dietary K,%			
	0	1.0	0	.73	1.3	1.8	Shade (30.1°C)[c]	No shade (40.9°C)[c]
Plasma								
Na	3084	3001[+]	3045	3035	3057	3028	3089	2996*
K	269	264	270	263[+]	265	268	263	270
Milk								
Na	549	515*	542	522	541	523	521	543
K	1927	1932	1934	1925	1915	1944	2005	1853**

[a] Least squares means.

[b] Dietary main effects pooled over environments.

[c] Black globe temperatures 1300 to 1500 h the last 14 days of each experimental period.

[+] Signifies difference between pairs of means, $P<.1$.

* Signifies difference between pairs of means, $P<.5$.

** Signifies difference between pairs of means, $P<.01$.

(Mallonee et al., 1985). Furthermore, blood pH was higher, urine pH tended to be higher and pCO_2 was lower in NS than S (Table 3-6). As blood pCO_2 decreased during respiratory alkalosis bicarbonate was secreted into the urine to preserve acid-base balance (Dale and Brody, 1954). Cations, especially Na, would be secreted with bicarbonate to maintain electrical neutrality, further decreasing plasma Na concentrations (Morris, 1979).

Milk Na content was lower with added dietary $NaHCO_3$ (Tables 3-9 and 3-10). Milk K concentration was not affected by either dietary Na source or quantity or K content of the diet. Sasser et al. (1966) showed that K concentration of milk remained relatively constant despite widely varying K intakes. Dennis et al. (1976) reported no change in concentration of milk K as dietary K ranged from .45 to .66% K. Potassium deprivation (.12% K) decreased milk K concentration (Mallonee, 1984).

In this study milk K was 7.6% lower in NS than S (Table 3-10). Rook and Wood (1959) reported cows without shade had lower milk K compared to cows under shade. Kamal et al. (1961) detected no changes in milk Na concentrations or Na to K ratio, but lower milk K content during exposure to high temperature. Decreased milk K content during heat stress suggests that ion is used for other higher priority physiological processes such as sweating. In our study milk Na concentrations were similar for the two environments.

Summary

Increased production responses to dietary Na and K concentrations above NRC (1978) requirements have been demonstrated in this and previous experiments (Mallonee, 1984; Mallonee et al., 1982; Chapter II). Diets

containing added $NaHCO_3$ or NaCl (each resulting in .55% total dietary Na, dry basis) and differing only in quantity of bicarbonate ion increased actual milk yields. Our results thus indicate that the Na moiety per se may contribute considerably to the milk yield responses often observed with $NaHCO_3$ feeding (Escobosa et al., 1984; Snyder et al., 1983; Erdman et al., 1982, 1980; Rogers et al., 1982; Chapter II). Additionally, 4% FCM yields (unadjusted for feed intake) increased 9.4% with added $NaHCO_3$ but only 4.0% with NaCl, indicating $NaHCO_3$ and specifically bicarbonate ion increased 4% FCM yield via increased milk fat content. Effect of added Na on 4% FCM yield did not change as dietary Na concentration increased from .55 to .88% indicating no additional need for Na in the diet above .55% (Figure 3-1). Milk yield increased as dietary K increased from 1.0% of diet to 1.5% (Chapter II) and in this experiment feed intake and actual milk yield (unadjusted for feed intake) increased as dietary K increased from 1.3 to 1.8% of diet. Since K content of milk is higher than any other mineral, lower dietary K may limit production. Furthermore, supplementation of these mineral salts increased rate of passage of digesta and feed intake (Haaland and Tyrrell, 1982; Rogers et al., 1982; Chalupa, 1979; Harrison et al., 1975) possibly influencing assimilation of nutrients and thus production responses. Severity of heat stress in NS and associated physiological responses to heat stress (Table 3-6) may explain interaction of environment with dietary K on milk yield. Although response to dietary treatments generally was greater in S than NS, S nevertheless represented a degree of heat stress and benefits to feeding higher dietary concentrations of Na and K in general were apparent in both environments.

CHAPTER IV
NYCTEROHEMERAL PATTERNS OF ACID-BASE STATUS, MINERAL METABOLISM AND DIGESTIVE FUNCTION OF LACTATING COWS IN NATURAL OR CHAMBER HEAT STRESS ENVIRONMENT

Introduction

A variety of effects of heat stress on production responses and general health, physiology and welfare of dairy cattle have been reviewed (Collier et al., 1982; Thatcher and Collier, 1982; Thatcher, 1974; McDowell, 1972). Many intensive studies focusing on acid-base measurements, mineral balances or other metabolic parameters have utilized climatic chambers. In most cases, animals were maintained in constant, nonfluctuating hot environments. Many basic studies done at the University of Missouri investigating effects of heat stress on acid-base balance (Dale and Brody, 1954), mineral metabolism (Kamal et al., 1962) as well as on other aspects of cattle physiology were conducted in climatic chambers at constant temperatures (Warren et al., 1974; Johnson et al., 1967, 1966; Kelly et al., 1967; Kibler et al., 1967; Wayman et al., 1962; Brody et al., 1948; Ragsdale et al., 1948). Absence of nycterohemeral patterns of ambient temperatures, as well as solar radiation and air movement make it difficult to compare studies conducted in natural and chamber environments. For example, under natural conditions cattle are able to radiate heat accumulated during the day to the nighttime sky, this is impossible in a chamber environment.

Digestibilities of feed components increase (NRC, 1981; Warren et al., 1974; Wayman et al., 1962; Davis and Merilan, 1960) and concentrations of volatile fatty acids (VFA) (Warner, 1981; Mertens, 1977; Johnson, 1970) decrease during heat stress. When feed intake was maintained constant in hot and thermoneutral environments, VFA concentrations were lower in hot environments as were amplitude and frequency of ruminal contractions (Niles et al., 1980; Gengler et al., 1970; Atterby and Johnson, 1968). Slower rates of passage of digesta and higher mean retention times have been hypothesized to account for changes in ruminal digestibility coefficients during heat stress (Wayman et al., 1962). In one study (Warren et al., 1974), mean retention time was 18% higher in steers at 32 compared to 18°C, though the higher temperature was constant over 26 h and feed intake was equal between temperatures. Actual rates of passage of solid and liquid phases have not been measured in lactating dairy cows during heat stress.

Objectives of this research were to compare acid-base balance, mineral metabolism and rates of passage of digesta through the rumen in dairy cows experiencing hyperthermia during a nycterohemeral time frame where daytime and nighttime ambient black globe temperatures were programmed to simulate natural heat stress conditions. Capability of chamber to simulate natural environment was determined using respiration rates, rectal temperatures and blood gases as indices of comparison.

Materials and Methods

Two heat stress studies were conducted; an experiment under near natural conditions (shade management system (S) vs no shade (NS)) (Roman-Ponce et al., 1977) and a climatic chamber experiment (thermoneutral (TN) vs heat stress (HS) environment).

Shade/No Shade Management Model Experiment (Natural Environment)

Twelve early to mid-lactation Holstein cows were assigned randomly to either shade or no shade environments during the summer at the Dairy Research Unit, Hague, FL (29°44' N latitude, 82°26' W longitude). Cows were maintained in respective environments for 35 days prior to sampling. Complete mixed diet consisted of about 40% corn silage and 60% ground corn-based concentrate mix (Table 4-1). Cows were milked at 0600 and 1800 h and were fed at 1000 and 1800 h daily. Drinking water was available continuously. Hourly blood samples were taken from 1300 h for 26 consecutive hours by jugular vein catheter. Blood was sampled in glass syringes, immediately capped, iced and analyzed for blood pH and gases by a semiautomated system (Corning Medical 165/2 Blood Gas Analyzer, Medfield, MA). In addition black globe temperature (BGT) (Buffington et al., 1981), data for black globe-humidity index (BGHI) (Buffington et al., 1981), respiration rates, rectal temperatures and urine for urine pH were obtained hourly. Black globe-humidity index incorporates effects of dry bulb temperature, net radiation, air movement and humidity into one index which is related directly to rectal temperature, respiration rate and milk production (Buffington et al., 1981).

Table 4-1. Formulation of diets for shade/no shade and chamber experiments.

Shade/No Shade Experiment		Chamber Experiment	
Ingredient	% dry matter	Ingredient	% dry matter
Corn silage	37.9	Alfalfa haylage	39.5
Ground corn	41.4	Ground corn	54.4
Soybean meal	16.5	Soybean meal	5.8
Limestone	.85	Trace mineral sodium chloride	.28
Dicalcium phosphate	.30	Vit. A,D,E premix[b]	.06
Vit. A,D,E premix[a]	.90		
Magnesium oxide	.21		
Trace mineral sodium chloride	.25		
Ammonium chloride	1.50		
Potassium chloride	.19		

[a] Provided 4000 IU vitamin A, 2500 IU vitamin D, and 20 IU vitamin E/kg diet dry matter.

[b] Provided 3400 IU vitamin A, 3100 IU vitamin D, and 20 IU vitamin E/kg diet dry matter.

Chamber Heat Stress Environment

Two environmental chambers, each containing two cows, were used to simulate natural nycterohemeral pattern of ambient black globe-humidity indices. Under these controlled conditions fluctuating weather conditions were not a factor and more critical animal measurements were possible. Hourly and nycterohemeral sequences of BGT were controlled by microprocessor. One chamber was programmed to simulate BGT of a typical Florida summer day without shade (HS) while the other was maintained at a constant thermoneutral environment (Figure 4-1). Chambers are unable to fabricate natural radiation, therefore, relative humidity was maximized during day hours of HS environment to produce the level of stress that solar radiation would produce in the natural environment. Nycterohemeral pattern of percent relative humidity paralleled BGT and ranged from 82% during the day (0900 to 2100 h) to 70% at night (2200 to 0800 h). Cows were milked in the chambers at 0600 and 1800 h and feeding was at 1000 and 1800 h daily. Complete mixed diets consisted of 40% alfalfa haylage and 60% ground corn based concentrate mix (Table 4-2). Individual cow feed and water (cups with in-line flow meters) intakes were measured daily. Animals were ruminally fistulated and the costoabdominal artery, located on the caudal side of the last rib, was catheterized (Guilbault, 1984).

Experimental design of chamber study was a single reversal. In each period, two animals were adapted to respective environments for days 1 through 10 followed by a sampling period, days 11 through 17. Animals were then adapted to the alternate environment (10 days)

Figure 4-1. Effect of natural shade (+——) or no shade (□---) and chamber thermoneutral (+——) or heat stress (□---) environments on black globe humidity index, respiration rates and rectal temperatures measured over 26 h. Shaded areas signify cool hours of the day.

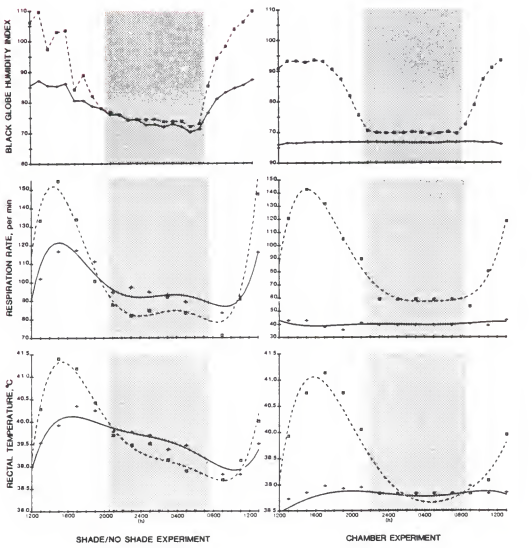

BLACK GLOBE HUMIDITY INDEX
RESPIRATION RATE, per min
RECTAL TEMPERATURE, °C
1200
1600
2000
2400
0400
0800
1200
(h)
SHADE/NO SHADE EXPERIMENT
CHAMBER EXPERIMENT

Table 4-2. Laboratory analyses of diets.

Component	Shade/No Shade Experiment	Chamber Experiment
	% dry matter	
Crude protein	18.54	13.6
Acid detergent fiber	15.30	22.7
Calcium	.97	.36
Phosphorus	.37	.36
Magnesium	.29	.20
Potassium	1.30	1.30
Sodium	.18	.16
NE_1^a, Mcal/kg	1.69	1.47

[a] Estimated from formulation.

followed by a second 7-day sampling period. On day 11 of each sampling period, ruminal ingesta, blood and urine samples were taken hourly from 1200 h for 26 hours. Ruminal contents were squeezed through 4 layers of cheese cloth to obtain ruminal fluid. Liquid dilution rates from the rumen were measured by disappearance of chromium. Thirty-five grams of LiCr-EDTA crystal, prepared according to (Uden et al., 1980), were dissolved in 1 liter of water and dosed through the fistula at 1000 h. Samples were taken hourly from each cow starting at 1200 h on day 11 of each sampling period.

Rates of passage of solids were measured by appearance of ytterbium (Yb) in feces. Ytterbium marked fiber was prepared in the following manner. Undigested fiber was separated from feces collected from cows fed the basal diet prior to the actual experiment by washing over a screen (Hydrasieve Static Screen, Bauer Bros. Co., Springfield, OH). Fiber was rinsed thoroughly, soaked overnight in a commercial detergent, thoroughly rinsed in water, soaked for 1 h in acetone and then dried at 65°C. Fiber (500 g) was then immersed in a solution of 100 g $YbCl_3 \cdot 6H_2O$ (Research Chemicals, Phoenix, AZ) in 10 liter of water for 48 h. After thorough rinsing, Yb-marked fiber was dried at 65°C. Total amount of marked-fiber needed was prepared prior to initiation of experiment. Compared to other methods of marking fiber (i.e., spraying), immersion is most preferred as it allows for maximum binding (Mader et al., 1984). Immediately prior to feeding, 500 g of marked-fiber was dosed into the rumen through the cannula. About 3.34 grams of Yb was administered to each cow and fecal concentrations reached over 500 ppm. Teeter et al. (1984)

suggests that marker dosages should be sufficient to achieve a minimum of 200 ppm Yb in the digesta samples obtained.

Fecal samples were taken at 6 h intervals on days 11 through 16 and at 12 hour intervals on day 17. Ellis et al. (1983) and Hartnell and Satter (1979) have shown that turnover rates estimated from appearance of marker in feces were similar to rates of Yb disappearance from the rumen and a reliable measurement to determine rates of passage of ruminal solids.

Ytterbium was extracted from fecal samples dried at 65°C according to method of Hart and Polan (1984). Ten ml of .05 M EDTA acid solution in .05% KCl (pH adjusted to 6.5 with ammonium hydroxide) were added to samples in 100 ml centrifuge tubes with screw caps and shaken for 30 minutes. Chromium and Yb concentrations were analyzed using atomic absorption spectrophotometry with a nitrous oxide flame (Model 5000, Perkin-Elmer Inc., Norwalk, CT).

Arterial blood for gas analysis was sampled every hour in glass syringes, capped and immediately analyzed. Separate plasma samples were obtained by centrifugation at 3000 x g for 20 min. Creatinine was measured in blood plasma and urine by colorimetric method using .04 M picric acid (Henry et al., 1974). Sodium and K from plasma, urine and ruminal fluid were measured by atomic absorption spectrophotometry (Model 5000, Perkin-Elmer Inc., Norwalk, CT). Osmolality of plasma and ruminal fluid were measured with a vapor pressure osmometer (Model 510013, Wescor Inc., Logan, UT). Urine ammonium was determined by semiautomated method (Technicon Autoanalyzer Model II,

Terrytown, NY) measuring absorbance of ammonia-salicylate complex at 660 nm (Hambelton, 1977).

In both experiments differences in nycterohemeral profiles between environments were tested for by homogeneity of regression using the general linear model procedures of SAS (SAS, 1982). Differences between environmental means pooled across time were tested by method of least squares analysis of variance using general linear model procedures (SAS, 1982). Results of all tests of homogeneity of regression are found in Tables 4-3 and 4-4.

These experiments were designed to study response variables as a function of time. Although differences between means were tested, power of statistical analysis for testing means was not great due to low number of experimental animals. Nevertheless, attention is often called to large differences despite lack of statistical significance.

Results and Discussion

Comparison of Hot Environments

Production responses. Cows in the heat stress chamber environment consumed less feed (13.6 vs 18.4 kg/d; $P<.01$), more water (81.9 vs 86.0 liter/d; $P<.01$) and produced less milk (16.5 vs 20.0 kg/d; $P<.01$) compared to thermoneutral environment. These results are comparable with previous research in the S/NS management model (Chapters II and III; Mallonee et al., 1985).

Acid-base physiology. Diurnal patterns of acid-base status were measured under natural environmental conditions of the shade/no shade

Table 4-3. Degrees of freedom and results of tests of significance for homogeneity of pooled versus separate regression for environmental treatments in shade/no shade experiment where variables were measured over 26 hours.

Item	df in F test[a]	Signif.
Respiration rate	5,261	**
Rectal temperature	5,265	**
Blood pH	3,246	NS
Blood pCO_2	5,242	**
Blood HCO_3	5,242	*
Hematocrit	4,268	**
Plasma protein	4,279	**
Urine pH	4,279	**

* $P<.05$

** $P<.01$

[a] Numerator of F test is mean square when a single pooled regression is fitted. Degrees of freedom of numerator correspond to order of polynomial that best fits data. Denominator of F test is residual mean square when separate regressions are fitted for each treatment.

Table 4-4. Degrees of freedom and results of tests of significance for homogeneity of pooled versus separate regressions for environmental treatments in chamber experiment where variables were measured over 26 hours.

Item	df in F test[a]	Signif.
Respiration rate	5,192	**
Rectal temperature	5,194	**
Blood pH	5,190	**
Blood pCO_2	5,190	**
Blood HCO_3	5,190	*
Blood pO_2	5,190	**
Plasma osmolality	5,90	NS
Urine NH_4^+/Urine creatinine	2,83	**
Urine pH	4,194	**
Plasma potassium	b	
Plasma sodium	b	
Plasma creatinine	2,83	NS
Urine creatinine	1,86	NS
Urine K/Urine creatinine	1,84	NS
Urine Na/Urine creatinine	1,84	NS
Rumen pH	4,187	**
Rumen osmolality	5,91	*
Total ruminal volatile fatty acids	5,91	NS
Molar percent acetate	b	
Molar percent propionate	2,94	NS
Molar percent butyrate	4,92	**

* P<.05

** P<.01

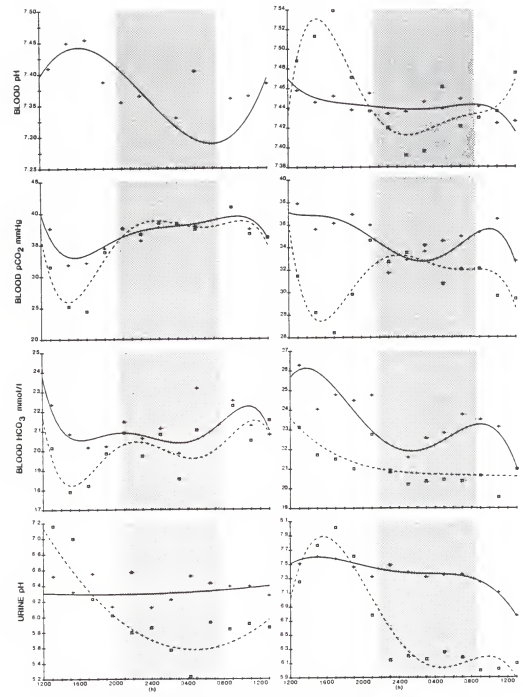
[a] Numerator of F test is mean square when a single pooled regression is fitted. Degrees of freedom of numerator correspond to order of polynomial that best fits data. Denominator of F test is residual mean square when separate regressions are fitted for each treatment.

[b] Regressions to fifth order not significant.

management model and in environmental chambers simulating BGHI of the natural environment (Figures 4-1 and 4-2). Shaded area in figures represents cool hours of day when BGHI, respiration rates and rectal temperatures were near basal levels. Maximum BGT was about 49°C in NS compared to about 37°C in HS. Nevertheless, rectal temperatures and respiration rates were similar between NS and HS environments (Figure 4-1). This is because percent relative humidities in chamber HS environment were kept high during hot hours to maximize stress. Maximum BGHI was about 109 in NS and about 94 in HS (Figure 4-1). Rectal temperatures for heat-stressed cows during hottest hours were 41.4°C in NS and 41.1°C in HS (Figure 4-1). Maximum respiration rates were about 155 per minute in NS compared to 143 per minute in HS. Under natural conditions, respiration rates respond more to solar radiation than any other meteorological factor (Bianca, 1965; Brody et al., 1948).

Shade structure under natural conditions blocked some of the solar radiation and effectively lowered body heat load (Buffington et al., 1981). Despite the shade BGHI reached about 87 during the day and followed a natural 24 h pattern. Rectal temperatures and respiration rates in the natural S environment followed a circadian pattern and indicated some degree of heat stress during the day. Thermoneutral environment in chambers was programmed to maintain a constant 21°C. Rectal temperatures and respiration rates for cows in this environment remained constant at about 38.7°C and 40 per minute, respectively.

Figure 4-2. Effect of natural shade (+ ——) or no shade (□ ---) and chamber thermoneutral (+ ——) or heat stress (□ ---) environments on blood pH, pCO_2, HCO_3 and urine pH measured over 26 h. Shaded areas signify cool hours of the day.


BLOOD pH
BLOOD pCO_2 mmHg
BLOOD HCO_3 mmol/l
URINE pH
1200
1600
2000
2400
(N)
0400
0800
1200
SHADE/NO SHADE EXPERIMENT
CHAMBER EXPERIMENT

Animals in natural and chamber environments exhibited similar trends in blood gas responses to thermoregulatory stress. In both experiments nycterohemeral patterns of blood pCO_2 were lower in NS and HS environments versus S or TN environments. Nycterohemeral blood bicarbonate patterns were similar to pCO_2. The changes in ratios of bicarbonate to CO_2 resulted in an increased blood pH (Houpt, 1982) during hotter hours versus cooler hours. Differences in blood pH between environments were not detected in the natural S/NS model as S cows were subjected to temperatures above their upper critical temperature (NRC, 1981). In the chamber study, cows subjected to heat stress had a higher blood pH during the hot hours compared to TN. Dale and Brody (1954) and Bianca and Findlay (1962) used the term respiratory alkalosis to describe reduced blood CO_2 combining capacity and higher blood pH. According to their definition, cows in our experiments were experiencing respiratory alkalosis during heat stress. There appeared to be a renal compensation to alkalosis during hot hours as indicated by increased urine pH (Figure 4-2), probably due to increased bicarbonate excretion into urine (Bianca, 1965). Urine pH has been shown to increase even during mild heat stress (Bianca and Findlay, 1962). The bicarbonate buffering mechanism, in which bicarbonate (HCO_3^-) and pCO_2 are relatively constant at a ratio of 20:1, is the most important chemical buffering system in the blood (Masero and Siegel, 1977). Thermally induced hyperventilation decreases pCO_2. To maintain the ratio at 20:1 and counter alkalosis, HCO_3^- is secreted by the kidney. This raises urine pH as was evident in these

experiments. Although the normal ratio between HCO_3^- and pCO_2 is maintained, amounts of each are subnormal. Correction is effected by renal excretion of H^+ which puts HCO_3^- back into the blood (Masero and Siegel, 1977). Lower urine pH and higher ammonium (Figure 4-3) concentration during the latter part of the cool hours indicated excretion of H^+. This suggests a large requirement and turnover of bicarbonate for buffering to maintain blood pH during heat stress. Overall nycterohemeral profiles of respiration rates, rectal temperatures and blood gas data indicate that heat stress in chambers was similar to natural heat stress in the S/NS management system. Means of blood gas measurements for the 26 hour sampling periods are in Table 4-5.

Nycterohemeral patterns of blood pO_2 were measured only in the chamber study (Figure 4-4). Animals in HS environment had pO_2 lower than TN during hours of hyperthermia, but 26 h means of pO_2 were not different. Others (Hales and Findlay, 1968; Bianca and Findlay, 1962) have measured higher blood pO_2 due to increased alveolar ventilation where oxen exhibited rapid shallow breathing during moderate heat stress or the slower deeper respiration seen in more severe heat stress where respiratory minute volumes reached seven times the control values.

Nycterohemeral pattern of blood hematocrit taken during the shade/no shade study suggested a slight hemo-concentration during the hot afternoon in the NS compared to S lot. Hematocrits were similar during the rest of the day between environments (Figure 4-5). Hematocrit means for the 26-hour sampling period were not

Figure 4-3. Effect of chamber thermoneutral (+ ——) or heat stress (□ ---) environments on urinary ammonium excretion presented as urine NH_4^+/urine creatinine measured over 26 h. Shaded area represents cool hours of the day.

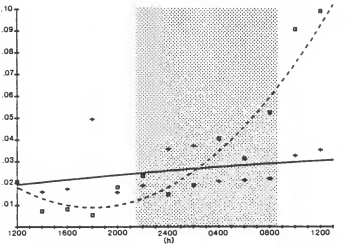

.10
.09
.08
.07
.06
.05
.04
.03
.02
.01
1200
1600
2000
2400
0400
0800
1200
(h)

Table 4-5. Environmental effects on daily means of physiological measurements.[a]

Item	Environment Model					
	Natural			Chamber		
	Shade	No shade	SE[e]	Thermo-neutral	Heat stress	SE[e]
Black globe humidity index	87	109		68	94	
Black globe temperature, °C[b]	34	49		21	37	
Rectal temp., °C	39.54	39.67	.37	38.83	39.55	1.03
Respiration rate, per min.	99.1	103.6	12.3	40.2	86.0**	4.1
Blood pCO_2, mmHg	36.68	34.87	2.41	34.94	31.28	6.79
Blood HCO_3, mmol/liter	21.22	20.06	2.29	23.45	21.24	4.98
Blood pH	7.379	7.333	.084	7.443	7.454	.037
Blood pO_2, mmHg	-	-		96.91	94.15	6.55
Hematocrit, %	28.0	28.1	1.8	-	-	
Plasma protein, %	7.64	7.18*	.413	-	-	
Plasma osmolality, mOsm/kg	-	-		284	285	8.9
Urine NH_4^+/ urine creatinine[d]	-	-		.026	.033	.009
Urine NH_4, mg/liter	-	-		59.48	109.03+	.01
Urine pH	6.32	5.99	.917	7.37	6.71*	.54
Fecal pH	-	-		5.65	5.67	.04
Plasma K, ppm	-	-		244	247	18.9
Plasma Na, ppm	-	-		2957	2899	67.4
Plasma creatinine, mg%	-	-		1.06	1.20+	.08
Urine creatinine, mg%	-	-		242	319	81
Urine K/Urine creatinine[c,d]	-	-		4.354	3.334	1.513
Urine Na/Urine creatinine[c,d]	-	-		.187	.094	.214

* P<.05

** P<.01

+ P<.10

[a] Least squares means of measurements taken of hourly intervals for 25 hours.

[b] Maximum daily temperature.

Table 4-5. continued.

[c] No nycterohemeral differences for plasma or urine creatinine across environments were detected. Data for urine K and Na are least squares means for the 25 hourly ratios of urine K or Na with urine creatinine (Kilmer et al., 1981).

[d] Expressed as (mg/100 ml)/(mg/100 mg creatinine)(Kilmer et al., 1981).

[e] Standard error of means.

Figure 4-4. Effect of chamber thermoneutral (+ ——) or heat stress (□ ---) environments on arterial blood pO_2 measured over 26 h. Shaded area represents cool hours of the day.

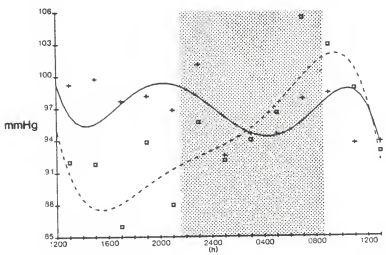

106
103
100
97
94
91
88
85
mmHg
1200
1600
2000
2400
0400
0800
1200
(h)

Figure 4-5. Effect of natural shade (+ ——) or no shade (□ ---) environments on percent hematocrit measured over 26 h. Shaded area represents cool hours of the day.

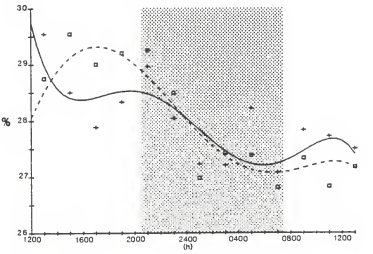
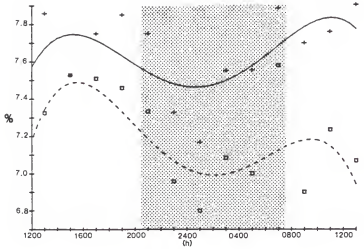

30
29
28
27
26
%
1200
1600
2000
2400
0400
0800
1200
(h)

Figure 4-6. Effect of natural shade (+——) or no shade (□ ---) environments on percent plasma protein measured over 26 h. Shaded area represents cool hours of the day.


7.8
7.6
7.4
%
7.2
7.0
6.8
1200
1600
2000
2400
0400
0800
1200
(h)

different between environments which agrees with previous reports (Chapter II; Buffington et al., 1981; El-Nouty et al., 1980). Unlike El-Nouty et al. (1980) plasma osmotic pressure was not different between environments (Tables 4-4 and 4-5).

In this experiment percent plasma protein was lower in the NS than S environment over the entire 26-hour sampling period which is in accord with (Collier et al., 1982; El-Nouty et al., 1980; McDowell et al., 1964; Kamal et al., 1962). This is due likely to an expanded plasma volume to accommodate increased evaporative water loss from the skin. This difference in percent plasma protein between environments appears to be smaller during the hot afternoon compared to the rest of the day. Hematocrit also suggested a hemo-concentration during the hot afternoon. Most likely cows in NS were too hot to expend energy in going to water which resulted in some dehydration. Others, however, did not detect effects of heat stress on percent plasma protein (Fuquay et al., 1980; Findlay, 1958).

Mean plasma and urine creatinine concentrations (Table 4-5) tended to be higher in HS versus TN chamber environments. This suggests catabolism of muscle resulting from reduced feed intake and agrees with (Thompson, 1973; Bianca, 1965; Findlay, 1958; Brody, 1956).

Creatinine is excreted at a constant rate in dairy cattle (DeGroot and Aefjes, 1960) and can serve as a reference to express rates of excretion of urinary metabolites when urine volume is unknown (Albin and Clanton, 1966; DeGroot and Aefjes, 1960). In this experiment secretion of urinary potassium per mg urinary creatinine

tended to be less in HS than TN environments probably due to increased potassium secretion in sweat (Mallonee et al., 1985; Johnson, 1970). El-Nouty et al. (1980) and Kamal et al. (1962) reported the same trend for urinary potassium excretion, however, they noted that urinary sodium output increased during heat stress. Our results indicate a trend towards a decrease in urinary sodium per mg urinary creatinine during heat stress.

Digestive Function

Ruminal turnover and dilution rates. Turnover rates of the solid phase of ruminal digesta can be determined by the appearance in the feces of a marked indigestible fiber that was dosed into the rumen (Ellis et al., 1983; Hartnell and Satter, 1979). Use of a potentially digestible material as a marker will result in estimation of turnover rate being confounded by rate of digestion (Ellis et al., 1979; Mertens, 1977). Fibrous material separated from feces can be assumed to be mostly lignified or undigestible since it has been exposed to the digestive capabilities of the ruminant. Dairy waste fiber was determined to be less than 20% digestible (Staples et al., 1981). These particles are of a specific size to enable escape from the rumen, therefore degradation into smaller particle size need not be incorporated into interpretation of passage rate data. Furthermore, materials high in fiber tend to have higher binding capacities for ytterbium (Ellis et al., 1982). Binding also renders material less digestible and since Yb is stable to digestive processes opportunity for marker migration is reduced (Teeter et al., 1984).

Effects of heat stress on rates of solids turnover and liquid phase dilution were measured in the chamber experiment. Ytterbium and chromium concentrations underwent natural logarithm transformation and were best fit to first order polynomial equations. According to Grovum and Williams (1973) use of first order kinetics to describe elimination of marker is of fundamental importance in developing a mathematical model. Homogeneity of regression indicated that liquid dilution rates were faster in the TN compared to the HS environment (Table 4-6). Testing marker concentrations as a function of time is a more powerful statistical test as the error term has many more degrees of freedom than a factorial arrangement of individual animal dilution rates. For example, in this experiment there were 194 df for error in regression analysis, whereas testing means of regression coefficients between environmental treatments had only 2 df in the error term. However, liquid dilution and solid turnover rates may be influenced by level of feed intake (Warner, 1981; Bull et al., 1979) making experiments with ad libitum consumption difficult to interpret. With one daily intake measurement covariance analysis cannot be used with tests for homogeneity of regression to reduce uncontrolled variation due to feed intake. Thus single estimate turnover rates were computed for individual animals and analyzed using analysis of variance with feed intake as covariate. Covariance analysis indicated no effect of feed intake or water intake on liquid dilution rate in this experiment and differences between environments were detected (Table 4-6).

Table 4-6. Environmental effect on rate of passage of liquid and solid digesta through the rumen (chamber study).

	Least squares means		Statistical Analysis				
	Thermo-neutral	Heat Stress	Homogeneity of Regression	Analysis of Variance	SE[b]	Analysis of Variance with feed intake as covariate[a]	SE[c]
Liquid dilution rate, %/h	10.9	8.6	**	*	.003		
Rumen volume, liter	38.3	89.5	d	NS	10.726		
Total rumen liquid outflow, liter/day	233.3	195.5	d	NS	25.807		
Solid turnover rate, %/h	3.6	2.8	**	*	.0014	+	.00014
Mean retention time, h	27.9	38.4	d	NS	3.237		

* P<.05

** P<.01

+ P<.06

[a] Effect of covariate statistically significant (P<.05).

[b] Standard error of means analyzed by analysis of variance.

[c] Standard error of means for solid turnover rate with feed intake as covariate.

[d] Tests for homogeneity of regression were not performed.

Differences in ruminal volume and total ruminal liquid outflow were not detected due to large between animal variation.

Homogeneity of regression indicated that rate of solid turnover was slower in the HS compared to the TN environment (Table 4-6). When solid turnover rates for individual animals were analyzed using analysis of variance with feed intake as covariate, HS animals had lower rates than TN ($P<.06$).

Ruminal non-kinetic measurements. Nycterohemeral patterns of ruminal pH measured in the chamber experiment indicated declines in pH in response to feeding (Figure 4-7). Although individual feeding patterns were not recorded, we observed that TN cows consumed large meals after fresh feed was offered at 1000 and 1800 h with resultant declines in ruminal pH. Heat-stressed cows ate when the temperature was cooler (2200 to 0900 h). Therefore, in heat-stressed cows during hot hours there was little rapidly fermentable substrate in the rumen that could lower pH. Decline in ruminal pH at about 2000 h reflects feeding and subsequent increase in ruminal acid production. Mean ruminal pH (Table 4-7) was not different between treatments. Contrary to our findings lower ruminal pH has been reported for heat-stressed cows (Niles et al., 1980; Bandaranayaka and Holmes, 1976; Mishra et al., 1969). This discrepancy might possibly be due to time of day samples were taken in relation to specific eating patterns during heat stress and to type of diets used.

Means of rumen osmolality (Table 4-7) did not differ between environments in the chamber study. Potassium and sodium

Figure 4-7. Effect of chamber thermoneutral (+ ——) or heat stress (□ ---) environments on ruminal pH measured over 26 h. Shaded area represents cool hours of the day. Cows were fed at 1800 and 1000 h daily.

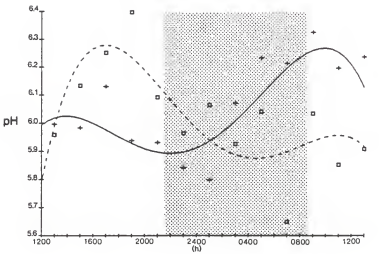

pH
6.4
6.3
6.2
6.1
6.0
5.9
5.8
5.7
5.6
1200
1600
2000
2400
0400
0800
1200
(h)

Table 4-7. Environmental effects on ruminal non-kinetic measurements in chamber study.[a]

	Thermoneutral	Heat Stress	SE
pH	6.05	6.03	.239
Osmolality, mOsm/kg	276	281	17.4
Sodium, ppm	2438	2210	445
Potassium, ppm	2204	2059	997
Total VFA, mMol/liter	119.0	108.2*	3.10
Acetate, molar%	62.0	61.9	.87
Propionate, molar%	27.4	26.6	1.40
Butyrate, molar%	10.5	11.5*	.58
Acetate:Propionate	2.46	2.46	.076

* $P<.05$

[a] Least squares means of measurements taken at hourly intervals for 26 h.

concentrations in ruminal fluid were not different between environments despite differences in feed intake.

Nycterohemeral patterns of total VFA's, acetate and propionate were not different between environments, however, mean of total VFA concentrations were lower in the heat stress environment (Table 4-7). Means of molar percentages of acetate and propionate were not different across environments although butyrate was higher in the hot environment. Lower concentrations of total VFA's in hot compared to cool environments have been reported (Kelly et al., 1967; Weldy et al., 1964) but, lower molar percentage of acetate reported by others was not detected in this experiment. The reason for lower total VFA concentrations is unclear. Kelly et al. (1967) maintained constant intake by feeding weighbacks via ruminal fistula and still measured lowered VFA concentrations in heat-stressed cows. Gengler et al. (1970) did not detect differences in rumen VFA concentrations despite increasing ruminal temperature by placement of a thermode in the rumen. Changes in rumen liquid dilution rates will alter ruminal fermentation (Rogers and Davis,1982a, b; Chalupa, 1977; Harrison et al., 1975). Total VFA concentrations decrease but percentages of acetate increase and percentages of propionate decrease as liquid dilution rate increases. In this experiment, total VFA concentrations decreased 9.1% with slower liquid dilution rate during heat stress, but percent acetate and propionate were not affected.

Summary

Importance of hourly sampling is apparent in this study, as certain responses to the environment were in evidence only during specific intervals of the day. A complete image of the daily stress and recovery pattern necessitates such a sampling regimen. Blood gas data indicated that heat-stressed animals experienced respiratory alkalosis with subsequent renal compensation. Decline of these signs of alkalosis concurrent with decline of ambient temperature emphasizes the robust physiology of dairy cows.

Rates of passage of solid and liquid digesta were slower in heat stress despite removal of differences in feed intake due to environment by covariate analysis. From this experiment it appears that depression in voluntary feed intake due to heat stress is not directly causative of reduced rate of passage of digesta through the rumen. Rumen function during heat stress is probably altered by the anterior pituitary's effects on basal metabolism via reduced growth and thyroid hormones (Thatcher and Collier, 1982; Bianca, 1965).

CHAPTER V
EFFECT OF SUPPLEMENTAL POTASSIUM AND SODIUM CHLORIDE SALTS ON RUMINAL TURNOVER RATES, ACID-BASE AND MINERAL STATUS DURING HEAT STRESS

Introduction

Lactating dairy cows reduce feed intake and milk production during heat stress as a strategies to maintain normal body temperatures. These responses reduce heat resulting from digestion and metabolism. Subsequent increased digestibility coefficients (Warren et al., 1974; McDowell et al., 1964), slower rate of passage of digesta (Chapter IV) and lowered concentrations of total volatile fatty acids (Chapter IV; Weldy et al., 1964; Kelly et al., 1967) have been measured.

Potter et al. (1972), Warner and Stacy (1972), Hemsley et al. (1975), Harrison et al. (1975), Thomson et al. (1978) in sheep, Kellaway et al. (1977) in calves, Rogers et al. (1979, 1982a, b) and Croom et al. (1982) in steers and dairy cows have shown that salts of saliva (mainly $NaHCO_3$) and mineral salts increase liquid dilution rates and in many cases, increase feed intake. Efficiency of fermentation is a function of liquid dilution rate. As liquid dilution rate increases, the growth of new microbes increases and the amount of substrate as a percent of total substrate needed for microbial maintenance decreases (Isaacson et al., 1975). Postulated increase in turnover rate of liquid and solid digesta of

heat-stressed cows fed high levels of mineral salts may increase flow of soluble nutrients and small feed particles to the small intestine allowing feed consumption and consequently milk production to increase.

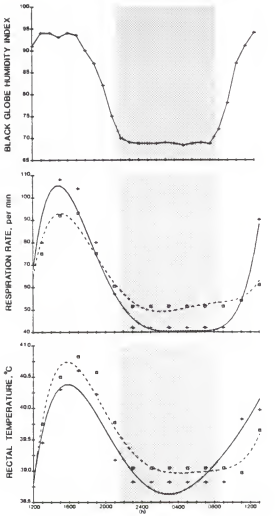
Sodium and potassium are alkalogenic elements (Cohen et al., 1972; Wheeler, 1981). High dietary levels of these cations fed to cows already challenged by a potential for environmentally induced respiratory alkalosis might further stress homeostatic mechanisms.

The objectives of this experiment were to study differences in ruminal liquid and solid turnover rates, acid-base balance and mineral metabolism of lactating dairy cows consuming either a basal or high mineral diet during heat stress within a nycterohemeral time frame.

Materials and Methods

Two environmental chambers, each containing two cows were used to produce thermal stress conditions. Under controlled chamber conditions natural weather fluctuation is not a factor and collection of precise data is possible. Hourly sequences of temperature and humidity were controlled by a microprocessor which was programmed to simulate black globe humidity indices (BGHI) (Buffington et al., 1981) typical of a Florida summer day (Figure 5-1). Black globe humidity index incorporates effects of dry bulb temperature, net radiation, air movement and humidity into one index which is related directly to rectal temperature, respiration rate and milk production. Nycterohemeral pattern of percent relative humidity

Figure 5-1. Circadian black globe humidity index pattern in chamber heat stress environments and effects of basal (+——) and high mineral (□---) diets on respiration rates and rectal temperatures measured over 26 h. Shaded area represents cool hours of the day.


BLACK GLOBE HUMIDITY INDEX
RESPIRATION RATE, per min
RECTAL TEMPERATURE, °C
1200
1600
2000
2400
0400
0800
1200
hrs

ranged from 82% during the day to 70% at night. Relative humidity was high during daytime hours in order to maintain the level of stress that solar radiation would have produced under natural conditions. Cows were milked in the chambers at 0600 and 1800 h, feeding was at 1000 and 1800 h daily. Basal diet (B) consisted of 50% corn silage and 50% ground corn and soybean meal based concentrate mix (Tables 5-1 and 5-2) fed as a total mixed ration. High mineral (HM) diet consisted of B plus 1.25% added NaCl and 1.85% added KCl. Individual cow feed and water (cups with in-line flow meters) intakes were measured daily. Cows were fistulated ruminally and the costoabdominal artery, located on the caudal side of the last rib, was catheterized (Guilbault, 1984).

Turnover rates of liquid phase from the rumen were measured by disappearance of chromium from the rumen. Thirty-five grams of LiCr-EDTA, prepared according to Uden et al. (1980), was dissolved in 1 liter of water and dosed through the rumen fistula at 1000 h. Twenty-six hourly samples were taken from each cow starting at 1200 each sample day.

Turnover rates of solid digesta were estimated by appearance of marker in the feces. Fecal fiber obtained from cows fed the basal diet during a preliminary period and marked with ytterbium was used as marker for solid phase digesta (Chapter IV). Ytterbium was extracted from fecal samples dried at 65°C according to the method of Hart and Polan (1984). Chromium and ytterbium concentrations were analyzed using atomic absorption spectrophotometry with a nitrous oxide flame (Model 5000, Perkin-Elmer Inc., Norwalk, CT).

Table 5-1. Formulation of experimental diets.

Item	Basal diet	High Mineral diet
	%	
Corn silage	50.0	50.0
Ground corn	33.3	30.2
Soybean meal	15.0	15.0
Trace mineral sodium chloride	.3	.3
Dicalcium phosphate	.3	.3
Magnesium oxide	.1	.1
Limestone	1.0	1.0
Sodium chloride	-	1.25
Potassium chloride	-	1.85

Table 5-2. Laboratory analysis of experimental diets.

Item	Basal diet	High Mineral diet
Crude protein, %	15.8	14.9
Acid detergent fiber, %	23.5	23.5
NE_l, (Mcal/kg)[a]	1.58	1.58
Calcium, %	.84	.78
Phosphorus, %	.43	.41
Magnesium, %	.31	.29
Potassium, %	1.50	2.30
Sodium, %	.15	.95
Chloride, %[b]	.20	1.85

[a] Estimated net energy for lactation.

[b] Calculated values for chloride.

Arterial blood for gas analysis was sampled hourly in glass syringes, capped and immediately analyzed by a semiautomated system (Corning Medical 165/2 Blood Gas Analyzer, Mayfield, MA). Plasma samples were obtained by centrifugation at 3000 x g for 20 minutes. Creatinine was measured in blood plasma and urine by colorimetric method using .04 M picric acid (Henry et al., 1975). Sodium and potassium concentrations in plasma, urine and ruminal fluid were measured by atomic absorption spectrophotometry. Osmotic pressure of plasma and ruminal fluid was measured with a vapor pressure osmometer (Model 510013, Wescor, Inc., Logan, Utah). Urine ammonium concentrations were determined by semiautomated method measuring absorbance of ammonia-salicylate complex at 660 nm (Hambelton, 1977).

Experimental design consisted of 2 periods in a single reversal. In each period, two cows were adapted to each diet for 10 days followed by a 7-day sampling period. Animals were then adapted to the other diet for 10 days followed by a second 7 day sampling period. Cows were subjected to the same nycterohemeral heat stress pattern for the entire experiment (17 days per period).

Differences in nycterohemeral profiles between dietary treatments were tested for by homogeneity of regression (Snedecor and Cochran, 1980) using general linear model procedures of SAS (SAS, 1982). Differences between treatment means were tested by method of least squares analysis of variance using general linear model procedures. Results of all tests for homogeneity of regression are found in Table 5-3. This experiment was designed to study response variables as a function of time. Although differences between means

Table 5-3. Degrees of freedom and results of tests of significance for homogeneity of pooled versus separate regressions for dietary treatments where variables were measured over 26 hours.

Item	df in F test[a]	Signif.
Rectal temperature	5,189	**
Respiration rate	5,189	**
Blood pH	2,197	NS
Blood pCO_2	3,195	NS
Blood HCO_3	3,195	NS
Blood pO_2	1,196	NS
Urine pH	3,196	**
Plasma K	4,89	*
Plasma Na	2,92	NS
Plasma osmolality	b	
Plasma creatinine	b	
Urine creatinine	4,90	NS
Urine K/urine creatinine	4,87	NS
Urine Na/urine creatinine	5,76	NS
Urine NH_4/urine creatinine	1,92	NS
Rumen pH	5,193	*
Rumen osmolality	3,88	NS
Rumen Na	4,91	NS
Rumen K	4,82	NS
Rumen total volatile fatty acids	5,91	NS
Molar percent acetate	b	
Molar percent propionate	b	
Molar percent butyrate	5,91	+

+ $P<.10$

* $P<.05$

** $P<.01$

Table 5-3. continued.

[a] Numerator of F test is mean square when a single pooled regression is fitted. Degrees of freedom of numerator correspond to order of polynomial that best fits data. Denominator of F test is residual mean square when separate regressions are fitted for each treatment.

[b] Regressions to fifth order not significant.

were tested, power of statistical analysis for testing means was not great due to low number of experimental animals. Nevertheless, attention was often called to large differences despite lack of statistical significance.

Results and Discussion

Cows receiving high mineral diet tended to consume more feed dry matter (21.0 vs 21.3 kg/d; NS), produce more milk (17.4 vs 18.3 kg/d; NS) and consume more water (70.23 vs 82.25 liter/d; $P<.01$) compared to cows receiving the basal diet. These results agree with earlier findings at this experiment station where heat-stressed cows had higher intakes and milk yields with higher levels of dietary sodium and potassium salts (Chapters II and III).

University of Florida environmental chambers have been shown to duplicate indices of thermal stress exhibited by cows in a natural heat stress environment (Chapter IV). Respiration rates, rectal temperatures and blood gas measurements were similar between the artificially created chamber environment and a typically natural heat stress environment. In this experiment, mean rectal temperatures ranged from 40.8°C during the hot hours to 38.9°C in the cool hours (Figure 5-1). Mean respiration rates ranged from 100 to 48 per minute (Figure 5-1). Shaded areas in figures represent cool hours of day when black globe humidity index, respiration rates and rectal temperatures were near basal levels.

Ruminal Kinetics

Ruminal turnover of liquid and solid digesta were analyzed using regression on marker concentration data which had undergone natural logarithm transformation versus time. Data from the descending phase of the fecal excretion curve were used to calculate solid turnover regressions. The shapes of the regression curves that best fit both liquid and solid data were second order polynomial equations. Comparison of regression lines of B versus HM diets for both liquid and solid markers indicated that these regression lines were not parallel suggesting responses to treatments differed over time. Equations for each is given in Table 5-4. Plotting of regression lines for liquid turnover suggests that HM tended to be faster compared to B. Plotting solids turnover regression lines for HM and B, however, yielded no clear indication of a difference in turnover rates despite the fact that the lines were not parallel.

Marker concentrations for each cow also were fit to straight lines in order to obtain regression coefficients (slopes). Analysis of variance with feed intake as covariate supported the above trend that liquid turnover rate tended to be faster ($P<.10$) for HM than B, but there was no difference in solids turnover rates. Difference in liquid turnover rates between these dietary treatments might have been greater had the K level of B been at the intended .8% NRC (1978) recommendation instead of the actual 1.5%.

Estimates of ruminal volume based on chromium dilution data were about 160 liters. This would seem too high, even though Adams et al. (1984) reported rumen volumes up to 149 liters in steers. This may

Table 5-4. Effects of dietary mineral level on turnover rates of liquid and solid digesta through the rumen during heat stress.

	Regression equation	%/h[c]	SE[d]
Liquid turnover			
Basal diet[a]	$= 3.382-0.14568x_1+.00225130x_2$**	8.5+	.0022
High mineral diet	$= 3.322-0.13924x_1+.00160260x_2$	9.5	
Solid turnover			
Basal diet[b]	$= 6.453-0.00190x_1-0.00036700x_2$**	4.6	.0034
High mineral diet	$= 7.121-0.03566x_1-0.00008271x_2$	4.7	

+ $P<.10$

** $P<.01$, 2 curves are not parallel

[a] Regression equations for 26 hourly samples of chromium turnover from rumen.

[b] Regression equations for appearance of Yb in feces sampled over 6 days.

[c] Regression coefficients (slopes) when curves are fit to straight lines.

[d] Standard error between each pair of means of regression coefficients for liquid and solid turnover rates.

have occurred since markers were dosed immediately prior to feeding. Feeding increases liquid dilution rate and rumen volume (Stokes et al., 1985; Warner and Stacy, 1968). Furthermore, the volume estimates were based on linear regression coefficients when in fact the data better fit second order curves. Nycterohemeral temperature pattern likely caused differences within daily turnover rates suggesting large deviations from steady state within each 24-hour period. Rapid turnover rates after eating resulting in curvilinear marker dilution curves, have been shown in sheep dosed immediately prior to eating (Ulyatt, 1964) and in dairy cows dosed 5 h prior to eating (Stokes et al., 1985).

Influence of HM diet to increase liquid turnover rate is further supported by effects on VFA patterns (Table 5-5). Cows consuming HM diet had lower concentrations of total VFA, with trends towards higher molar percentages of acetate, lower molar proportion of propionate and higher acetate to propionate ratio. This agrees with other reports (Chalupa, 1977; Rogers et al., 1982) where feeding higher levels of dietary minerals increased liquid turnover rates and affected VFA patterns.

Mineral and Acid-Base Metabolism

High potassium and sodium contents of HM diet suggest it would exert an alkalogenic effect on acid-base status (Wheeler, 1981). Average potassium intake for cows on HM diet was 490 g/day. However, cows fed pasture or green-cut alfalfa regularly may consume 500 g or more of potassium (Ward, 1966). Anderson and Pickering (1962) reported that K excretion in urine of cows increases to equal the rate of intravenous infusion of up to 6 to 7 meq/liter with only a

Table 5-5. Effect of dietary mineral level on ruminal non-kinetic measurements during heat-stress.[a]

Item	Basal diet	High Mineral diet	SE[b]
pH	6.26	6.30	.234
Osmolality, mOsm/kg	260	269**	1.561
Sodium, ppm	2212	2201	24
Potassium, ppm	1811	2294	550
Total volatile fatty acids, mmol/liter	97.34	80.29*	7.259
Acetate, molar %	65.77	68.71	5.611
Propionate, molar %	23.54	20.78	5.803
Butyrate, molar %	10.69	10.51	1.194
Acetate:Propionate	2.91	3.34	.818

* P<.05

** P<.01

[a] Least squares means of measurements taken hourly for 26 hours.

[b] Standard error for means.

1 to 2 meq/liter increase in plasma K concentrations. This suggests that the normally large turnover of K in the cow is associated with a large excretory reserve capacity with excretion of alkaline urine ($KHCO_3$) but without upsetting acid-base status (Pickering, 1965).

National Research Council (1978) recommends about .3% chloride (Cl) as part of a total NaCl requirement. The ARC (1980) estimates that about 61.6 g Cl is required daily for a 600 kg cow producing 40 kg milk (Coppock, 1978). Average daily Cl consumption, about 394 g for the HM diet, was in excess of these recommendations in the current experiment.

Chloride exerts an acidogenic effect on acid-base physiology (Cohen, 1973). An association has been shown between excess dietary Cl^- and acidosis for poultry and swine (Leach, 1979; Yen et al., 1981). Therefore, a potential for metabolic acidosis on HM diet existed. Compensation to high acidogenic load is evident in blood gas and urine data.

According to the Henderson-Hasselbach equation, blood pH, of which the bicarbonate buffering system is the principal buffer, is determined by the ratio of base (HCO_3) to acid (pCO_2) (Houpt, 1982). As dietary acidogenic factors react with blood bicarbonate, pCO_2 increases which causes respiratory control systems to stimulate respiration rate to restore the balance of HCO_3 to pCO_2. This may explain the higher respiration rates exhibited during cooler hours (Figure 5-1) and the lower blood HCO_3 (Table 5-6) of cows consuming HM compared to B diet. Acidemia also causes secretion of H^+ by renal tubular cells, mostly as ammonium ions (Scott, 1969a), which alleviates the acid load and restores bicarbonate to the plasma.

Table 5-6. Effects of dietary mineral level on daily means of rectal temperature, respiration rate, blood gases and mineral metabolism during circadian heat stress.[a]

Item	Basal diet	High Mineral diet	SE[c]
Rectal temperature, °C	39.42	39.54	1.25
Respiration rate, per min	61	63	17.9
Blood pCO_2, mmHg	34.64	33.44	3.43
Blood HCO_3, mmol/liter	24.06	22.25*	.93
Blood pH	7.459	7.437	.028
Blood pO_2, mmHg	90.27	92.24	2.77
Urine pH	7.78	7.15+	.60
Fecal pH	6.18	6.26	.03
Plasma K, ppm	253	261	11.5
Plasma Na, ppm	3109	3042	177.6
Plasma osmolality, mOsm/kg	282	286	4.4
Plasma creatinine, mg%	1.27	1.24	.27
Urine creatinine, mg%	184.8	113.9	100.5
Urine K/urine creatinine[b]	7.25	10.92	6.47
Urine Na/urine creatinine[b]	.327	.398	.396
Urine NH_4^+, mg/liter	26.3	35.9	.013
Urine NH_4^+/urine creatinine[b]	.022	.034	.003

* $P<.05$

+ $P<.10$

[a] Least squares means of measurements taken at hourly intervals for 26 hours.

[b] Expressed as (mg/100 ml)/(mg/100 mg creatinine).

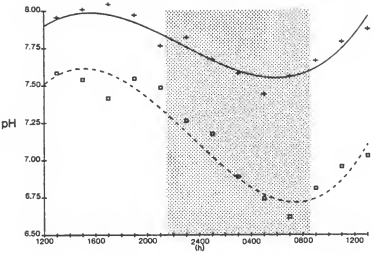
[c] Standard error of means.

Renal response to acidosis was not influenced by K excretion in sheep (Scott, 1969b).

Confounded with the dietary effects of HM diet on acid-base status in this experiment is the hyperthermic environment. Previous research with dairy cows experiencing a similar pattern of heat stress (Chapter IV) indicated significant trends towards respiratory alkalosis during hot hours of the day. In this experiment cows on both diets had lower pCO_2 and HCO_3 than normal indicative of respiratory alkalosis (Kronfeld, 1975). Furthermore, pCO_2 and blood pH tended to be lower and HCO_3 was lower for HM compared to B diet (Table 5-6) in agreement with the above description of response to metabolic acidosis. Nycterohemeral pattern of urine pH (Figure 5-2) indicates renal compensation for respiratory alkalosis during hot hours of the day for both dietary treatments when respiration rates were high compared to cooler hours. Urine pH was higher as bicarbonate secretion compensated for the heat-induced alkalosis (Bianca, 1965). Urine pH for cows on HM diet was consistently lower than B throughout the nycterohemeral time frame and mean urinary ammonium concentrations tended to be higher (Table 5-6) suggesting response to the acidogenic nature of the diet. Escobosa et al. (1984) fed lactating dairy cows 1.67% chloride during heat stress and reported lowered blood pH, pCO_2, HCO_3 and urine pH for treatment compared to control cows which agrees with our findings.

Consumption of HM diet resulted in a slightly higher ruminal osmolality compared to B (Table 5-5). Changes in ruminal osmolality after meals are due to increases in concentrations of ammonia, VFA

Figure 5-2. Effect of chamber heat stress environment, and basal (+ ——) or high mineral (□ ---) diet on urine pH measured over 26 h. Shaded area represents cool hours of the day.


8.00
7.75
7.50
pH 7.25
7.00
6.75
6.50
1200
1600
2000
2400
0400
0800
1200
(h)

and K (Bennink et al., 1978; Scott, 1975; Warner and Stacy, 1972a, 1972b). For both diets in this experiment, ruminal osmolality was close to 260 mOsm/kg, the optimum for ciliates (Durand et al., 1980). Ruminal K concentrations tended to be higher, but ruminal sodium concentrations were not increased by dietary treatment. This is consistent with other observations of K and Na concentrations in rumens of sheep and steers (Greene et al., 1983a, 1983b; Newton et al., 1972; Scott, 1975; Warner and Stacy, 1972b). At high ruminal K concentrations Na absorption from the rumen increased (Warner and Stacy, 1972a, 1972b) but at low K concentrations rumen Na concentrations increased due to slower rate of absorption from the rumen. With increased rumen Na concentrations lower urinary Na was measured suggesting aldosterone release in response to Na retention in the rumen. The absorption mechanism is likely a NaK-ATPase in the rumen epithelium (Warner and Stacy, 1972b) that can be disabled by oubain (Harrison and Munn, 1970). Potassium-facilitated Na absorption may explain why rumen Na concentration did not increase on the HM diet (Table 5-5). Sodium absorption is the major mechanism maintaining the resting rumen isotonic to blood (Warner and Stacy, 1972). This might explain why the increase in rumen osmolality was small (although significant) with HM diet. Requirements for the maintenance of the ionic environment of the rumen dominate systemic regulation of electrolytes and fluid (Gans and Mercer, 1982; Stacy and Warner, 1966).

Urinary K tended to be higher with HM diet, however, no differences between treatments for plasma K, or plasma or urinary Na

were detected (Table 5-6). Increases in plasma and/or urinary K without change in plasma Na have been demonstrated for both cattle and sheep fed high K diets (St. Omer and Roberts, 1967; Deetz et al., 1982; Newton et al., 1972; Scott, 1969a; Greene et al., 1983; Erdman et al., 1980; Escobosa et al., 1984) and with high Na diets (Hemsley et al., 1975). Plasma Na concentrations are regulated closely since plasma osmolality and blood volume are dependent upon Na concentrations more than any other osmotic determinant. Potassium secretion, however, is usually proportional to plasma concentration and membrane potential across luminal membrane of renal tubular cells (Pitts, 1975). Elevated loads of filtered K can increase urine volume due to decreased water reabsorption in the proximal tubule and loops of Henle (osmotic diuresis) (Deetz et al., 1982). Reabsorption of Na may be decreased due to concentration gradient favoring net diffusion of Na from interstitial fluid to lumen (Vander, 1980). Higher urinary Na has been reported with sheep and steers fed high K diets (Newton et al., 1972; Warner and Stacy, 1972; Greene et al., 1983a, b). St. Omer and Roberts (1967) reported no increase in urinary Na concentrations with varying dietary K levels (157 to 1087 mEq) fed to heifers.

Summary

Feed intake and milk yield were increased during heat stress by the addition of mineral salts to the diets of lactating cows (Chapters II and III). Furthermore, slower dilution rates of liquid and solid digesta in the rumen were quantified in heat-stressed cows

compared to cows in thermal neutrality (Chapter IV). Objectives of the present experiment were to determine if the addition of mineral salts to the diets of heat-stressed cows would increase rates of passage of liquid and solid digesta through the rumen. Although actual difference between dietary mineral levels was smaller than intended there was a measurable increase in liquid turnover rate. Turnover of solids was not affected in this experiment. The trend toward increased feed intake and milk yield and the significant alteration in VFA fermentation patterns, compatible with increased liquid turnover rate, gives further evidence that the HM diet increased turnover of liquid digesta through the rumen.

CHAPTER VI
GENERAL RESULTS AND DISCUSSION

Earlier research at the University of Florida has shown increased production responses when dietary levels of Na and K were higher than the NRC (1978) recommended levels (Mallonee et al., 1985). Increasing dietary K, especially during warm months, can be justified for three reasons. Potassium secretion via milk can account for 15 to 40% of total daily K intake. High environmental temperatures may increase dietary requirement due to increased loss via sweating (Jenkinson and Mabon, 1973; Mallonee et al., 1985). Many feedstuffs, especially concentrates and by-product feeds, are low in K. When feed intake is depressed during heat stress, increased K intake may be required. Since Na and K are closely interrelated and reciprocate ionically in many physiological functions, increased Na intake might be warranted as well.

Lowered ruminal pH has been reported in heat-stressed cows (Niles et al., 1980; Bandaranayaka and Holmes, 1976). It is hypothesized that this may be due to CO_2 being blown off during panting which would upset the HCO_3/CO_2 ratio. The resulting tendency for respiratory alkalosis would be countered by HCO_3 excretion by the kidney. Loss of CO_2 could possibly deplete the bicarbonate substrate pool available for salivary buffering of the rumen. Heat-stressed cows may be fed high proportions of concentrates to increase the

energy density of their diet and high concentrate diets may lower ruminal pH as well (Dirkson, 1970). Supplementing rations, especially high energy rations, with ruminal buffers during heat stress was explored in this research. Usage of $NaHCO_3$ and/or $KCHO_3$ would have the advantage of supplying the buffer and the mineral together.

In experiments 1 and 2, cows were kept in either S or NS lots as described by Roman-Ponce et al. (1977). They concluded that shade alters the microenvironment, lowers respiration rates, rectal temperatures and improves milk yield and reproductive performance compared to NS. In our experiments, rectal temperatures and respiration rates were lower in S versus NS.

There were definite tendencies for cows in NS to show signs of respiratory alkalosis compared to cows in S. These included higher blood pH, lower pCO_2 and HCO_3. Nevertheless, the average black globe temperature in S for both experiments was near the upper critical temperature for lactating dairy cows and respiration rates were elevated sufficiently to alter blood gases toward respiratory alkalosis. Although respiration rate is correlated strongly with solar radiation (Bianca, 1966) air temperature under the shade in the S environment was still high. This might explain the lack of difference in milk yield between environments in experiment 2.

In experiment 1, feed intake and milk yield were lower in NS compared to S for daytime measurements and for total daily measurements. In experiment 2, differences in feed intake were detected between environments, but not for milk yield.

Supplementation of $NaHCO_3$ increased milk yield in both experiments and improved percent milk fat in experiment 2. Milk fat was not measured in experiment 1 due to technical problems. Improvement in performance from $NaHCO_3$ may have been due to the effect of the buffer on ruminal pH. Attempts were made to measure ruminal pH via stomach tube but due to the large amounts of saliva generated during the procedure, attempts were terminated.

Supplementation of $KHCO_3$ at 1% of dietary dry matter in experiment 1 had a unexpected effect on feed intake and milk yield. Other reports in the literature (i.e., Emery and Brown, 1961) did not report intake problems with $KHCO_3$. In experiment 1, total daily feed intake was lower and daytime, nighttime and total daily milk yield were lower when $KHCO_3$ was fed. In experiment 1, the roughage source was cottonseed hulls. If the problem with $KHCO_3$ was due to palatability, cottonseed hulls most likely would not have masked any unpleasant flavors.

There were two levels of total dietary K in experiment 1, 1.0 and 1.5%. There was no effect of K level on total feed intake, but milk yield was higher with 1.5% K. In half of the 1.5% K diets, the source of supplemental K was KCl and in the other half, the source was $KHCO_3$. This may explain why intake did not improve with the higher K level as was seen with Mallonee et al. (1985) who fed only KCl. Intake was greater in this experiment when KCl versus $KHCO_3$ was the source of supplemental K.

In experiment 2, dietary K levels were 1.3 and 1.8% with KCl as the only source of supplemental K. Both feed intake and milk yield

were higher at 1.8 versus 1.3% K. When feed intake across dietary treatments was equalized by including intake in the statistical model as a continuous independent variable, there were no effects on milk yield due to K. This suggests that the K effects on milk yield are via feed intake since removing the effect of K on feed intake also removes its effect on milk yield.

The beneficial effects of $NaHCO_3$ in experiment 1 may have been due to the Na cation and its possible effects on digesta turnover rates, physiological interactions with K or supplementation of a subclinical Na deficiency. The effects of $NaHCO_3$ also may have been due to the bicarbonate buffer. In order to further understand the effects of Na and HCO_3, NaCl was used also as a dietary treatment in experiment 2. Sources of Na could thus be compared. Feed intake and milk yield were not affected by addition of NaCl. But, when differences due to feed intake were equalized statistically, milk yield and 4% fat-corrected milk were higher with NaCl. The reasons for this are unclear, although the data suggest that response to NaCl was for reasons other than an increase in feed intake.

There was a NaCl by $NaHCO_3$ interaction for both feed intake, milk yield and 4% fat-corrected milk. When neither supplement was fed and total dietary Na was .18% of dry matter intake (the NRC recommended level), both feed intake and milk yield were lower than when either NaCl or $NaHCO_3$ were fed with total dietary Na at .55%. Supplementation of diet with both NaCl and $NaHCO_3$ (.88% total dietary Na), however, did not improve either feed intake or milk yield above the .55% Na level.

Increased production responses to dietary Na and K concentrations above the NRC (1978) recommendation have been demonstrated in these and previous experiments (Mallonee et al., 1985). Diets containing NaCl or $NaHCO_3$ in experiment 2 and differing only in the anion increased milk yield by about 5.0%. In experiment 1, $NaHCO_3$ increased milk yield by 7.2%.

These results indicate that the Na moiety per se contributes to the milk yield response often observed with feeding $NaHCO_3$ (Erdman et al., 1980; Rogers et al., 1982; Snyder et al., 1983). Futhermore, 4% fat-corrected milk increased 9.3% with $NaHCO_3$, but only 4.6% with NaCl suggesting that the bicarbonate ion specifically increased percent milk fat.

The above results for dietary treatments hold for both S and NS environments as no environmental by dietary treatment interactions were detected except for dietary K in experiment 2. Milk production in S increased 8.5% with 1.8% compared to 1.3% dietary K, whereas no effect of K was noted in NS. Although response to dietary treatments generally was greater in S than NS, S nevertheless represented a degree of heat stress and benefits to feeding higher dietary concentrations of Na and K generally were apparent in both environments.

There was little effect of mineral salt treatments on acid-base physiology. Sodium bicarbonate did not effect blood pH in either experiment although blood HCO_3, total CO_2 and urine pH were higher in experiment 2. In the first experiment, blood pH was higher and pCO_2 lower with $KHCO_3$. In experiment 2, NaCl supplementation raised blood

HCO_3 and total CO_2 but not blood pH. Feeding 1.8 versus 1.3% dietary K raised blood and urine pH. This is likely due to the alkalogenic effect of the Na and K ion (Cohen, 1972).

In both experiments, plasma Na was lower in NS than S but plasma K was not different. Reports of lower plasma concentrations of aldosterone (El-Nouty et al., 1980; Israel et al., 1978) would suggest less of a need to conserve Na and more of a need to conserve K (Guyton, 1976) since Na and K (in relation to H ions) are reciprocally excreted or reabsorbed at the distal tubules of the kidney (Masoro and Siegel, 1971). This is likely a strategy to conserve K for other high-priority physiological needs such as neural and muscular functions and loss in sweat during hyperthermia. Conservation of K during heat stress suggests increased renal requirement for Na due to the reciprocal Na excretion and K reabsorption mechanism. Lower plasma Na supports this concept of increased Na excretion by the kidney.

Plasma volume may be increased during heat stress (El-Nouty et al., 1980) which would dilute the absolute quantity of plasma Na. In these experiments, however, there was no effect of environment on hematocrit. Furthermore, as bicarbonate secretion into the urine increased during heat stress, cations, especially Na, are secreted with HCO_3 to maintain electrical neutrality further decreasing plasma Na concentrations.

Sodium is the major determinant of plasma osmolality and thus blood volume (Guyton, 1976). It is regulated closely by various osmo- and volume receptors in the body. The kidney can very

precisely maintain body Na levels. Therefore, detectable differences in plasma Na concentration would certainly result only from significant stimuli.

Experiments 3 and 4 were designed to answer questions raised in experiments 1 and 2. For example, how do ruminal kinetics change during heat stress and what is the mechanism whereby higher mineral salts in the diet increase feed intake and milk yield? Do they increase rate of passage of digesta as has been reported elsewhere under thermoneutral conditions? Actual rates of passage of digesta have not been measured during heat stress. We also were interested in further characterizing effects of heat stress on acid-base balance, especially over a 24-hour period.

Ability of the environmental chambers at the University of Florida to elicit in lactating dairy cows the same indices of thermal stress, i.e., respiration rates, rectal temperatures and blood gas variables, as are exhibited under natural conditions was verified in experiment 3. In both natural and chamber studies, cows were in either control or heat stress environments. Overall circadian profiles of these variables were similar in the chamber and natural heat stress environments. Rectal temperatures for cows in heat stress reached over 40.0°C during hot hours in both natural and chamber situations and respiration rates were over 143 per minute. Blood pCO_2 and HCO_3 patterns for cows in natural and chamber heat stress environments were lower during hours of heat stress and rose toward more normal values as the temperature went down later in the day. Urinary pH tended to be higher during hot hours compared to

control as bicarbonate apparently was secreted by the kidney to counter respiratory alkalosis. During cool hours there was a decrease in urine pH and increase in NH_4^+ excretion. This probably was a result of a HCO_3 deficit accumulated during the day from increased CO_2 exhalation and secretion of HCO_3 into the urine. During the cool hours of the day, HCO_3 was put back into the blood with resultant excretion of H^+ into the urine.

It had been hypothesized that loss of HCO_3 substrate by increased respiration rate and increased urinary HCO_3 secretion would decrease the pool available for salivary buffers. However, in experiment 3, nycterohemeral pattern of ruminal pH indicated a higher pH during the hot hours of the day instead of the lower values which had been hypothesized and daily means for ruminal pH were not different across environments. This suggests that despite altered acid-base status, salivary buffering capacity was sufficient to maintain ruminal pH. Intake patterns influence ruminal pH since pH tended to decrease after animals consumed feed.

The nycterohemeral profile of pO_2 indicated lower partial pressure for heat-stressed cows during the hours of heat stress. A possible correlation between lowered pO_2, altered pCO_2 and HCO_3 and slower gut smooth muscle activity resulting in decreased rate of passage of digesta might warrant further investigation.

Blood plasma protein measured in the S/NS study indicated a hemo-dilution during the hot hours in the NS environment. This probably is due to expanded plasma volume for increased surface heat dissipation.

In experiment 4, four cows were subjected to heat stress in the environmental chambers and fed either a basal or high mineral diet (1.25% NaCl and 1.85% KCl added to the basal diet). Sodium and K are alkalogenic elements (Cohen et al., 1972), but effects of acid-base balance due to the intake of these cations was not detected. Urine K excretion was, however, higher for cows consuming high mineral diet. The anion, chloride, was supplied by both supplemental salts. Chloride apparently did affect acid-base balance as was in evidence by lower blood HCO_3, slightly lowered blood pH and lower urinary pH.

The benefits to frequent sampling over 24 h was made clear in these chamber studies since certain responses were observable only during specific parts of the day. Responses to heat stress concurrent with actual temperature changes would not have been detected otherwise.

In experiment 3, turnover rates of liquid and solid digesta through the rumen were compared for heat-stressed cows versus thermoneutral cows. Turnover rates for both liquid and solid digesta through the rumen were slower in heat stress even when variations due to feed intake were equalized statistically. Concentrations of total volatile fatty acids were lower for heat stressed cows which agrees with previous reports (Kelly et al., 1967; Weldy et al., 1964).

Based on these findings, experiment 4 was designed to attempt to increase turnover rate of liquid and solid digesta of heat-stressed cows by increasing the mineral content of the diet. There was a small but measurable increase in liquid turnover rate, however,

turnover of solids was not altered by dietary mineral level. This may be due to the higher than intended mineral level of the basal diet which was supposed to be at the NRC (1978) recommended level for Na and K. The trend toward increased feed intake and milk yield and the difference in volatile fatty acid fermentation pattern which occurred with the high mineral diet are compatible with increased liquid turnover rates and yield further support that the high mineral diet increased turnover of liquid digesta through the rumen.

This research has indicated a need for higher mineral levels for lactating dairy cows during the heat stress conditions that are found in the southeastern United States and other subtropical and tropical parts of the world. Directly related to the decrease in feed intake consistently observed by hyperthermic cows is a decrease in rate of passage of digesta through the rumen. Whether this is a result of decreased feed intake or a result of other factors such as the influence of the hypothalamus, changes in thyroid or other hormone levels or due to the upset in blood gases warrants further investigation. However, the potential for improving productive performance under actual commercial conditions was demonstrated by the increased liquid turnover rate resulting from the feeding of higher levels of Na and K salts. Furthermore, benefits of ruminal buffers, especially $NaHCO_3$ in stimulating feed intake, milk yield and percent milk fat observed in this research can be applied readily to field conditions. Finally, although homeothermic strategies employed by dairy cows are economically detrimental from the viewpoint of profits during summer, these strategies are enormously successful for

the actual survival of these animals under natural hot conditions. Attempts to increase production despite the animal's natural tendency to lower production during heat stress is not likely to overwhelm the cows capability to deal with stress.

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APPENDIX A
HOMOGENEITY OF REGRESSION ANALYSIS FOR CHAPTER IV

Table A-1. Tests for homogeneity of pooled versus separate regression for environmental treatments in shade/no shade experiment where variables were measured over 26 hours.

	Source	Respiration Rate df	Respiration Rate MS	Rectal Temperature df	Rectal Temperature MS	Blood pCO_2 df	Blood pCO_2 MS	Blood HCO_3 df	Blood HCO_3 MS	Blood pH df	Blood pH MS
Reduced Model[a] (partial SS) (sequential SS)	Shade	1	1213.45	1	1.2058	1	220.128	1	80.445	1	.1462
	Cow(shade)	10	1812.73	10	1.6502	10	69.464	10	62.643	10	.0846
	Hour	1	20077.13	1	49.2595	1	1189.461	1	27.941	1	.4820
	$Hour^2$	1	36648.07	1	.0811	1	192.063	1	40.334	1	.1996
	$Hour^3$	1	16314.88	1	33.0500	1	620.312	1	18.832	1	.4010
	$Hour^4$	1	558.12	1	3.7269	1	276.811	1	9.929		
	$Hour^5$	1	19589.92	1	12.4602	1	383.676	1	74.952		
	Residual(reduced)	266	242.37	270	.2152	247	9.788	247	3.034	249	.0853
	Residual(complete)	261	172.43	265	.1236	242	7.870	242	2.936	246	.0854
MSE Reduced-Complete[b]		5	3893.09	5	5.07	5	13.040	5	7.797	3	.146
F		5,261	**	5,265	**	2,242	**	5,242	*	3,246	NS
	Shade	1	1429.78	1	1.1929	1	221.682+	1	86.747	1	.1452
	Cow(shade)[a]	10	1820.60**	10	1.6319**	10	68.808**	10	62.444**	10	.0858
	Hour	23	4317.84**	23	4.4238**	22	140.055**	22	11.456**	22	.1251
	Shade*Hour	23	1058.53**	23	1.2685**	22	27.443**	22	3.097	22	.0882
	Residual	225	157.25	229	.1111	208	6.835	208	2.894	208	.0848

* P<.05

** P<.01

+ P<.10

[a] One regression line fit to both treatments.

[b] Separate regression lines fit to each treatment.

Table A-1. continued.

	Source	Blood pO_2 df	Blood pO_2 MS	Blood Hematocrit df	Blood Hematocrit MS	Plasma Protein df	Plasma Protein MS	Urine pH ds	Urine pH MS
Reduced Model[a] (partial SS) (sequential SS)	Shade	1	148.548	1	.0956	1	15.5476*	1	7.391
	Cow(shade)	10	71.272	10	36.5618	10	2.0458	10	10.097
	Hour	1	214.140	1	111.2574	1	.5681	1	7.293
	$Hour^2$	1	273.995	1	.8114	1	1.3286	1	6.484
	$Hour^3$	1	445.497	1	15.3014	1	.1882		
	$Hour^4$	1	218.728	1	.2786	1	2.2311		
	$Hour^5$			1	5.7317				
	Residual(reduced)	248	18.353	273	.8825	283	.0758	265	.303
	Residual(complete)	244	14.896	268	.8216	279	.0704	263	.248
MSE Reduced-Complete[b]		4	229.280	5	4.1420	4	.4520		
F		4,244	**	4,268	**	4,279	**	4,279	**
	Shade	1	134.165	1	.2328	1	15.585*	1	6.697
	Cow(shade)[a]	10	70.852	10	35.3678**	10	2.034**	10	10.249
	Hour	22	77.329	24	7.1198**	25	.453**	23	.784
	Shade*Hour	22	62.744	24	1.5722**	25	.130**	23	.803
	Residual	208	11.816	230	.7196	237	.047	221	.261

Table A-2. Tests for homogeneity of pooled versus separate regression for environmental treatments in chamber experiment where variables were measured over 26 hours.

	Source	Rectal Temperature df	Rectal Temperature MS	Respiration Rate df	Respiration Rate MS	Blood pCO_2 df	Blood pCO_2 MS	Blood HCO_3 df	Blood HCO_3 MS	Blood pH df	Blood pH MS
Reduced Model[a] (partial SS)	Chamber	1	.0028	1	2448.571	1	474.307	1	118.433	1	.01133
	Cow(chamber)	2	1.6763	2	5224.652	2	275.833	2	68.242	2	.00815
	Period	1	12.0690	1	1036.181	1	.371	1	47.093	1	.03160
	Treatment	1	27.3574	1	108481.462**	1	627.030	1	235.353	1	.00514
MSE Reduced[b] - MSE Reduced[a]	Pooled Interaction	2	4.2510	2	67.932	2	184.582	2	99.001	1	.00534
Reduced[b] Model (sequential SS)	Hour	1	5.4117	1	15045.156	1	19.314	1	172.896	1	.04198
	$Hour^2$	1	3.1796	1	13862.637	1	1.450	1	52.788	1	.01719
	$Hour^3$	1	1.6541	1	11055.918	1	65.528	1	5.889	1	.00529
	$Hour^4$	1	.9014	1	2475.519	1	5.469	1	19.246	1	.01872
	$Hour^5$	1	.5459	1	2773.635	1	107.130	1	16.925	1	.00464
	Residual	194	.2773	192	322.427	190	8.438	190	2.541	1	.00109
MSE Reduced[a] - Complete[c] F		5	7.0968	5	9288.332	5	88.733	5	7.370	5	.01297
		5,194	**	5,192	**	5,190	**	5,190	*	5,190	**
	Chamber	1	.0038	1	2471.707	1	510.638	1	123.748	1	.01291
	Cow(chamber)[b]	2	1.6906**	2	5314.912**	2	273.922**	2	68.090**	2	.00807**
	Period	1	11.9795**	1	1109.599**	1	.724	1	49.756**	1	.03196**
	Treatment	1	27.2006**	1	106788.806**	1	578.695**	1	222.281**	1	.00424**
	Hour	25	1.5661**	25	1934.607**	25	12.513	25	13.850**	25	.00407**
	Period*Hour	25	.0675	25	101.608	24	5.867	24	3.949	24	.00114
	Treatment*Hour	25	1.5398**	25	1914.846**	25	24.698**	25	3.401	25	.00301**
	Residual	126	.1402	124	66.270	123	11.993	123	3.445	123	.00080

* P<.05

** P<.01

[a] One regression line fit to both treatments.

[b] Cow, period and treatment are absorbed in reduced model.

[c] Separate regression lines fit to each treatment.

Table A-2. continued.

	Source	Blood pO_2 df	Blood pO_2 MS	Urine NH_A df	Urine NH_A MS	Urine NH_4^+/ Urine creatinine df	Urine NH_4^+/ Urine creatinine MS	Urine pH df	Urine pH MS	Fecal pH df	Fecal pH MS	Plasma Potassium df	Plasma Potassium MS
Reduced Model[a] (partial SS)	Chamber	1	58.2115	1	.02039	1	.00168257	1	10.9974	1	.00056	1	473.3168
	Cow(chamber)	2	116.2455	2	.03228	2	.00233145	2	1.8108	2	.08744	2	1049.9968
	Period	1	572.1047	1	.00296	1	.00340469	1	8.1108	1	.00124	1	1165.7427
	Treatment	1	372.3311	1	.06936	1	.00078790	1	23.1087*	1	.01678	1	191.6163
MSE Reduced[b]-MSE Reduced[a]	Pooled Interaction	2	171.7900	2	.00492	2	.00029800	2	1.1819			2	1428.6170
Reduced[b] Model (sequential SS)	$Hour_2$	1	538.1096	1	.26542	1	.02289846	1	32.4569				
	$Hour_3$	1	1.9366	1	.04595	1	.00260615	1	.0649				
	$Hour_4$	1	200.9312					1	1.6733				
	$Hour_5$	1	27.8890					1	4.1665				
	Residual	1	453.2631										
		190	40.4985	90	.00466	83	.00043529	194	.1772	2	.00526		
MSE Reduced[a]-Complete[c] F		5	251.0310	2	.08958	2	.00591920	4	3.9908				
		5,190	**	2,90	**	2,83	**	4,194	**				
	Chamber	1	62.1655	1	.02967	1	.00149322	1	11.1078			1	415.5276
	Cow(chamber)	2	105.6701*	2	.02247**	2	.00039010	2	1.8184			2	947.1718*
	Period	1	528.4857**	1	.00488	1	.00489301	1	8.2128			1	1300.4384*
	Treatment	1	388.4306**	1	.05994**	1	.00095452	1	22.7410			1	188.0450
	Hour	25	85.7007**	12	.02616**	12	.00180390	25	1.6052			12	160.8411
	Period*Hour	24	73.2351**	12	.00233	12	.00048130	25	.1544			12	329.0078
	Treatment*Hour	25	62.3710**	12	.01753**	12	.00146940	25	.7454			12	240.4029
	Residual	123	30.9270	58	.00327	59	.00026115	125	.0945			60	203.4079

Table A-2. continued.

	Source	Plasma Sodium df	Plasma Sodium MS	Plasma Osmotic Pressure df	Plasma Osmotic Pressure MS	Plasma Creatinine df	Plasma Creatinine MS	Urine Creatinine df	Urine Creatinine MS	Urine K/Urine Creatinine df	Urine K/Urine Creatinine MS	Urine Na/Urine Creatinine df	Urine Na/Urine Creatinine MS
Reduced Model[a] (partial SS)	Chamber	1	177632.9864	1	162.657	1	.0989	1	7745.84	1	6910.351	1	1.8776
	Cow(chamber)	2	5239.3355	2	1.956	2	.2497	2	91982.48	2	2091.236	2	12.8851
	Period	1	7536.6924	1	1040.884	1	.1529	1	69234.99	1	639.538	1	7.3225
	Treatment	1	72174.4441	1	69.157	1	.3861	1	127751.67	1	2212.271	1	21.4422
MSE Reduced[b]-MSE Reduced[a]	Pooled Interaction	2	18149.000	2	318.143	2	.0273	2	26362.40	2	915.99	2	18.327
Reduced[b] Model (sequential SS)	$Hour_2$			1	10.740	1	.4770	1	152641.44	1	8016.156	1	33.4291
	$Hour_3$			1	13.145	1	.5154						
	$Hour_4$			1	14.489								
	$Hour_5$			1	.036								
	Residual			1	2.770								
				90	44.575	83	.0569	86	4472.38	84	135.371	84	1.2978
MSE Reduced[a]-Complete[c] F				5	84.291	2	.0012	1	6181.26	1	239.008	1	1.3166
				5,90	NS	2,83	NS	1,86	NS	1,84	NS	,184	NS
	Chamber	1	158720.000*	1	167.387*	1	.1081	1	10005.78	1	7281.708	1	2.9469
	Cow(chamber)	2	2144.464	2	2.118	2	.2062*	2	86047.56**	2	1860.256**	2	14.1712**
	Period	1	15706.871	1	1049.316**	1	.1021	1	74876.63**	1	520.145	1	7.2760
	Treatment	1	82280.077	1	65.189	1	.3848*	1	133141.55**	1	2271.612**	1	19.1740**
	Hour	12	65139.519	12	23.865	12	.0673	12	15583.74**	12	773.551**	12	3.0455
	Period*Hour	12	77895.401	12	23.891	12	.1130*	12	3978.26	12	60.636	12	.7718
	Treatment*Hour	12	17635.730	12	54.145	12	.0117	12	6540.08	12	88.175	12	.9776
	Residual	59	66694.510	61	56.734	51	.0581	53	5088.66	51	190.687	51	2.2684

Table A-3. Tests for homogeneity of pooled versus separate regression for environmental treatments in chamber experiment where variables were measured over 26 hours.

		Rumen											
		Rumen pH		Osmotic Pressure		Rumen Sodium		Rumen Potassium		Total VFA		Molar % Acetate	
	Source	df	MS	df	MS	df	MS	df	MS	df	MS	df	MS
Reduced Model[a] (partial SS)	Chamber	1	.70487	1	1712.346	1	1865392.45	1	6252259.11	1	.0094	1	197.8926
	Cow(chamber)	2	.61845	2	2184.500	2	4948.70	2	101570.85	2	794.2742	2	785.0047
	Period	1	.01129	1	1929.846	1	9929.78	1	2421466.27	1	494.9396	1	42.3278
	Treatment	1	.03589	1	572.462	1	1377729.08	1	310895.81	1	3034.0242*	1	.8033
MSE Reduced[b] -MSE Reduced[a]	Pooled Interaction	2	.229	2	1205.692	2	791380.45	2	3976981.0	2	38.3845	2	3.0520
Reduced[b] Model (sequential SS)	$Hour_2$	1	.0061	1	18.473	1	43219.05			1	2079.5057		
	$Hour_2$	1	.15893	1	166.264	1	88923.46			1	2275.5985		
	$Hour^3$	1	.09607	1	1755.148	1	24257.49			1	2351.1243		
	$Hour^4$	1	.62793	1	212.298	1	757592.07			1	2357.3789		
	$Hour^5$			1	4206.807					1	2320.3986		
	Residual	187	.06838	92	246.916	90	53614.21			91	150.1126		
MSE Reduced[a] -Complete[c] F		4	.68054	5	685.662	4	30575.23			5	270.1640		
		4,187	**	5,91	*	4,90	NS			5,91	NS		
	Chamber	1	.66787	1	1712.346	1	1856771.98**	1	5602930.20*	1	.0094	1	197.8926
	Cow(chamber)	2	.60433**	2	2184.500**	2	5539.20	2	64558.38	2	794.2742**	2	785.0047**
	Period	1	.00776	1	1929.846**	1	14149.61	1	2025214.25**	1	494.9396	1	42.3278*
	Treatment	1	.03625	1	572.462	1	1317971.13**	1	440885.37	1	3034.0242**	1	0.8033
	Hour	25	.07608	12	686.237**	12	107603.62	12	183747.83	12	342.8337**	12	2.9430
	Period*Hour	23	.06030	12	137.888	12	70127.02	12	26974.24	12	89.7576	12	2.3955
	Treatment*Hour	25	.15893**	12	444.837	12	69391.65	12	78628.50	12	220.8436	12	8.1896
	Residual	120	.05727	62	258.099	60	70347.40	49	216240.75	62	1360.0719	62	11.0330

* P<.05

** P<.01

[a] One regression line fit to both treatments, pooled.

[b] Cow, period and treatment are absorbed in reduced model.

[c] Separate regression lines fit to each treatment.

Table A-3. continued.

	Source	Molar % Propionate df	Molar % Propionate MS	Molar % Butyrate df	Molar % Butyrate MS	Acetate:Propionate df	Acetate:Propionate MS
Reduced Model[a] (partial SS)	Chamber	1	131.0830	1	6.8554	1	6.20179
	Cow(chamber)	2	1033.5443	2	18.0545	2	19.34744
	Period	1	33.1380	1	.5616	1	.42795
	Treatment	1	18.7581	1	27.3249*	1	.00104
MSE Reduced[b]-MSE Reduced[a]	Pooled Interaction	2	7.8695	2	1.3307	2	.02288
Reduced[b] Model (sequential SS)	$Hour_2$	1	4.3305	1	.0355		
	$Hour_3$	1	28.0778	1	.9008		
	$Hour_4$			1	.1632		
	$Hour_5$			1	5.9231		
	Residual			92	.7940		
MSE Reduced[a]-Complete[c]		5	270.1640	2	25.9861	4	4.2078
F		2,94	NS	4,92	**		
	Chamber	1	131.0830	1	6.884	1	6.7018
	Cow(chamber)	2	1033.5413**	2	18.0545**	2	19.3475**
	Period	1	33.1380	1	0.5616	1	.4280
	Treatment	1	18.7581	1	27.3249**	1	.0010
	Hour	12	4.8416	12	.8349	12	.0463
	Period*Hour	12	2.9058	12	.3015	12	.0408
	Treatment*Hour	12	10.8973	12	1.5858*	12	.1373
	Residual	62	14.4629	62	0.8095	62	.2529

Table A-4. Tests for homogeneity of regression and analysis of variance for ruminal passage measurements.

	Source	In Rumen [Cr] df	In Rumen [Cr] MS	In Fecal [Yb] df	In Fecal [Yb] MS	Rumen Volume df	Rumen Volume MS	Mean Retention Time df	Mean Retention Time MS
Reduced Model[a] (partial SS)	Chamber	1	2.3518	1	7.4783				
	Cow(chamber)	2	17.9553	2	9.6223				
	Period	1	.0003	1	.2494				
	Treatment	1	5.5192	1	11.5918				
MSE Reduced[b] - MSE Reduced[a]	Pooled Interaction	2	1.8405	2	2.0370				
Reduced[b] Model (sequential SS)	$Hour_2$	1	107.5710	1	15.8680				
	$Hour_3$								
	$Hour_4$								
	$Hour_5$								
	Residual	195	.0481	124	.0951				
MSE Reduced[a] - Complete[c]		1	1.5569	1	2.608				
F		1,195	**	1,124	**				
	Chamber	1	.00021	1	.000120	1	24.5990	1	180.885
	Cow(chamber)	2	.00147*	2	.000028	2	89.4351	2	73.806
	Period	1	.00028	1	<.000001	1	580.0738	1	27.244
	Treatment	1	.00108*	1	.000153*	1	2.5645	1	220.223
	Residual	2	.00004	2	.000008	2	460.2085	2	41.922
	Chamber			1	.000069			1	78.460
	Cow(chamber)			2	.000006			2	18.629
	Period			1	.000001			1	3.935
	Treatment			1	.000003			1	3.798
	Feed Intake			1	.000016			1	83.489*
	Residual			1	<.000001			1	.354

* P<.05

** P<.01

[a] One regression line fit to both treatments.

[b] Cow, period and treatment are absorbed in reduced model.

[c] Separate regression lines fit to each treatment.

APPENDIX B
HOMOGENEITY OF REGRESSION ANALYSIS FOR CHAPTER V

Table B-1. Tests for homogeneity of pooled versus separate regressions for dietary treatments for variables measured over 26 hours.

	Source	Rectal temperature df	Rectal temperature MS	Respiration rate df	Respiration rate MS	Blood pCO_2 df	Blood pCO_2 MS	Blood HCO_3 df	Blood HCO_3 MS	Blood pH df	Blood pH MS
Reduced Model[a] (partial SS)	Chamber	1	5.209	1	1065.969	1	346.550	1	25.88	1	.0221
	Cow(chamber)	2	.040	2	727.627	2	325.995	2	137.39	2	.0004
	Period	1	.019	1	511.345	1	333.580	1	250.16	1	.0064
	Treatment	1	.696	1	121.105	1	74.100	1	169.77*	1	.0249
MSE Reduced[b] - MSE Reduced[a]	Pooled Interaction	2	6.252	2	1278.475	2	46.949	2	3.442	2	.0032
Reduced[b] Model (sequential SS)	Hour	1	19.341	1	8088.39	1	24.242	1	52.50	1	.0346
	$Hour^2$	1	14.257	1	8129.75	1	158.427	1	154.31	1	.0113
	$Hour^3$	1	21.459	1	6595.78	1	265.026	1	47.04		
	$Hour^4$	1	11.448	1	5355.03						
	$Hour^5$	1	2.185	1	4559.53						
	Residual	189	.258	189	134.89	195	6.204	195	3.905	197	.0012
MSE Reduced[a] - Complete[c] F		5	.966	5	973.53	3	3.663	3	1.778	2	.0028
		5,189	**	5,189	**	3,195	NS	3,195	NS	2,197	NS
	Chamber	1	.0017	1	2381.20	1	514.260	1	124.271	1	.0130
	Cow(chamber)	2	1.6906**	2	5314.91**	2	273.922**	2	68.090**	2	.0081**
	Period	1	11.9795**	1	1109.60**	1	.724	1	49.756**	1	.0320**
	Treatment	1	27.2006**	1	106788.81**	1	578.695**	1	222.281**	1	.0042*
	Hour	25	1.5661**	25	1934.61**	25	12.513	25	13.850**	25	.0041**
	Period*Hour	25	.0675	25	101.61	24	5.867	24	3.949	24	.0011
	Treatment*Hour	25	1.5393**	25	1914.85**	25	24.698**	25	3.401	25	.0030**
	Residual	126	.1402	124	66.27	123	11.993	123	3.445	123	.0008

* P<.05

** P<.01

[a] One regression line fit to both treatments.

[b] Cow, period and treatment are absorbed in reduced model.

[c] Separate regression lines fit to each treatment.

Table B-1. continued.

	Source	Blood pO_2 df	Blood pO_2 MS	Urine pH df	Urine pH MS	Fecal pH df	Fecal pH MS	Plasma Potassium df	Plasma Potassium MS	Plasma Sodium df	Plasma Sodium MS
Reduced Model[a] (partial SS)	Chamber	1	127.54	1	12.487	1	.00273	1	3589.114	1	54467.82
	Cow(chamber)	2	399.47	2	3.169	2	.00221	2	2033.913	2	305115.20
	Period	1	840.96	1	.457	1	.01208	1	80.540	1	115691.13
	Treatment	1	197.22	1	20.299	1	.01532	1	1777.398	1	116368.85
MSE Reduced[b]-MSE Reduced[a]	Pooled Interaction	2	30.60	2	1.436	2	.00406	2	529.575	2	126184.65
Reduced[b] Model (sequential SS)	Hour	1	468.44	1	8.050			1	1038.097	1	146112.40
	Hour 2			1	1.236			1	95.489	1	91927.97
	Hour 3			1	2.787			1	116.858		
	Hour 4							1	.234		
	Hour 5										
	Residual	196	38.05	196	.098			89	154.714	92	22372.95
MSE Reduced[a]-Complete[c] F		1	77.31	3	.659			4	446.892	2	34030.35
		2,197	NS	1,196	NS	3,196	**	4,89	*	2,92	NS
	Chamber	1	64.702	1	12.346	1	.0830	1	3757.160	1	60524.80
	Cow(chamber)	2	106.570	2	3.190**	2	.0400	2	2086.984**	2	279531.95**
	Period	1	528.486	1	.471*	1	.2562**	1	58.477	1	116228.26*
	Treatment	1	388.431	1	20.142**	1	.2979**	1	1644.146**	1	113185.40*
	Hour	25	85.701	25	.514**	22	.1608**	12	243.416	12	44192.12
	Period*Hour	24	73.235	25	.128	21	.1212**	12	139.789	12	16290.45
	Treatment*Hour	25	62.371	25	.127	22	.0335	12	246.412	12	19316.93
	Residual	123	30.927	126	.118	105	.0348	59	164.192	60	26814.90

Table B-1. continued.

	Source	Plasma Osmotic Pressure df	MS	Plasma Creatinine df	MS	Urine Creatinine df	MS	Urine K/Urine Creatinine df	MS	Urine Na/Urine Creatinine df	MS	Urine NH_4 df	MS	Urine NH_4/ Urine Creatinine df	MS
Reduced Model[a] (partial SS)	Chamber	1	54.086	1	.025844	1	127290.887	1	8233.311	1	61.1585	1	.00145	1	.0103260
	Cow(chamber)	2	21.702	2	.125474	2	3076.537	2	7639.291	2	79.2427	2	.00179	2	.0012363
	Period	1	127.163	1	.802423	1	26714.010	1	18745.128	1	10.3257	1	.00193	1	.0093187
	Treatment	1	380.779	1	.032788	1	122328.237	1	34915.712	1	21.0602	1	.00232	1	.0036947[NS]
MSE Reduced[b]-MSE Reduced[a]	Pooled Interaction	2	77.702	2	.294160	2	40404.910	2	16747.350	2	62.7470	2	.00069	2	.0027940
Reduced[b] Model (sequential SS)	$Hour_1$					1	30438.720	1	23014.293	1	114.7049	1	.01264	1	.0095435
	$Hour^2$					1	25571.399	1	15191.342	1	116.6925				
	$Hour^3$					1	5406.405	1	1634.350	1	47.2912				
	$Hour^4$					1	18991.289	1	13836.190	1	78.5536				
	$Hour^5$									1	59.1161				
	Residual					90	1388.500	87	1421.600	76	9.6607	94	.00032	92	.0011216
MSE Reduced[a]-Complete[c] F						4	749.720	4	1040.225	5	14.3605	1	.00166	1	.0001896
						4,90	NS	4,87	NS	5,76	NS	1,94	*	1,92	NS
	Chamber	1	54.087	1	.0317	1	123337.24*	1	7628.991	1	85.5705	1	.00139	1	.0099058
	Cow(chamber)	2	21.702	2	.1452*	2	3898.96	2	7690.226*	2	73.7625**	2	.00180	2	.0005949
	Period	1	127.164*	1	.8732**	1	23912.85**	1	16317.920*	1	11.5019	1	.00194	1	.0090304
	Treatment	1	380.779**	1	.0238	1	126596.55**	1	32266.495**	1	8.7164	1	.00234	1	.0035609
	Hour	12	33.864	12	.0260	12	8134.52**	12	4929.377*	12	37.9058*	12	.00127	12	.0017942
	Period*Hour	12	40.684	12	.0311	12	711.82	12	305.063	12	6.6673	12	.00018	12	.0008276
	Treatment*Hour	12	35.091	12	.0151	12	1028.58	12	968.060	12	10.8350	12	.00030	12	.0016285
	Residual	62	30.805	58	.0314	60	2810.60	57	2338.593	47	13.5267	61	.00038	59	.0013085

Table B-2. Tests for homogeneity of pooled versus separate regressions for dietary treatments for ruminal variables measured over 26 hours.

	Source	Rumen pH df	Rumen pH MS	Rumen Osmotic Pressure df	Rumen Osmotic Pressure MS	Rumen Sodium df	Rumen Sodium MS	Rumen Potassium df	Rumen Potassium MS	Total VFA df	Total VFA MS
Reduced Model[a] (partial SS)	Chamber	1	3.405	1	62.0805	1	43882.11	1	33200.99	1	282.947
	Cow(chamber)	2	2.503	2	714.0570	2	138392.72	2	1104845.70	2	424.599
	Period	1	.021	1	1076.3443	1	2070473.47	1	2135583.44	1	1060.942
	Treatment	1	.096	1	1773.3436**	1	477142	1	5726393.88	1	7555.863*
MSE Reduced[b] - MSE Reduced[a]	Pooled Interaction	2	.220	2	9.7495	2	2388.65	2	1210349.50	2	210.761
Reduced[b] Model (sequential SS)	Hour	1	.128	1	248.9988	1	206467.13	1	76744.86	1	418.678
	Hour2	1	.002	1	5.0569	1	489295.41	1	21143.73	1	24.494
	Hour3	1	.080	1	1869.1830	1	69807.36	1	73692.09	1	1189.680
	Hour4	1	.826			1	585419.87	1	1091889.13	1	20.014
	Hour5	1	.541							1	786.522
	Residual	193	.057	88	346.3730	91	32412.00	82	70395.94	91	124.084
MSE Reduced[a] - Complete[c] F		5,193	.1461 *	3,88	65.7920 NS	4,91	29552.98 NS	4,82	45924.5 NS	5,91	235.973 NS
	Chamber	1	3.521	1	101.8034	1	398.55547	1	7381.85	1	282.9473
	Cow(chamber)	2	2.516**	2	818.0140	2	141787.060*	2	1177359.80**	2	424.5990*
	Period	1	.033	1	1187.5899	1	2100404.264**	1	1766161.16**	1	1060.9424**
	Treatment	1	.081	1	2113.3318	1	3047.541	1	5151789.75**	1	7555.8625**
	Hour	25	.095*	12	225.2574	12	127371.880**	12	150213.55	12	256.7884*
	Period*Hour	25	.100*	12	712.9717	12	30909.784	12	99889.23	12	110.8841
	Treatment*Hour	25	.056	12	132.1832	12	14351.433	12	102583.47	12	158.0686
	Residual	125	.053	57	319.5207	61	36466.374	52	105177.13	62	126.5108

* P<.05

** P<.01

[a] One regression line fit to both treatments.

[b] Cow, period and treatment are absorbed in reduced model.

[c] Separate regression lines fit to each treatment.

Table B-2. continued.

	Source	Molar % Acetate df	Molar % Acetate MS	Acetate:Propionate df	Acetate:Propionate MS	Molar % Propionate df	Molar % Propionate MS	Molar % Butyrate df	Molar % Butyrate MS
Reduced Model[a] (partial SS)	Chamber	1	896.51524	1	18.991852	1	567.13424	1	37.5436
	Cow(chamber)	2	110.64691	2	.944765	2	22.65142	2	56.6172
	Period	1	5.10392	1	1.070519	1	93.43537	1	54.8638
	Treatment	1	224.48738	1	4.766455	1	197.97434	1	.8329
MSE Reduced[b] - MSE Reduced[a]	Pooled Interaction	2	125.94200	2	2.676850	2	134.69700	2	5.7058
Reduced[b] Model (sequential SS)	Hour							1	21.6429
	Hour*Treatment							1	.1199
	Hour^2							1	1.0106
	Hour^3							1	4.2692
	Hour^4							1	1.5689
	Hour^5							91	.8942
	Residual							5	1.9553
MSE Reduced[a] - Complete[c] F								5,91	P<.01
	Chamber	1	896.5152			1	567.1342*	1	37.5436
	Cow(chamber)	2	110.6469**			2	22.6514*	2	56.6172**
	Period	1	5.1039			1	93.4354**	1	54.8638**
	Treatment	1	224.4874**			1	197.9743**	1	.8328
	Hour	12	3.2922			12	1.2596	12	2.5946*
	Period*Hour	12	4.0430			12	2.1847	12	.8471
	Treatment*Hour	12	2.5080			12	2.1239	12	.9761
	Residual	62	6.5753			62	5.4524	62	1.1030

Table B-3. Tests for homogeneity of regression and analysis of variance for ruminal passage measurements.

	Source	ln Rumen [Cr] df	ln Rumen [Cr] MS	ln Fecal [Yb] df	ln Fecal [Yb] MS
Reduced Model[a] (partial SS)	Chamber	1	.7865	1	3.7238
	Cow(chamber)	2	5.8104	2	.4341
	Period	1	1.6722	1	.0001
	Treatment	1	.5205	1	.2705
MSE Reduced[b] -MSE Reduced[a]	Pooled Interaction	2	.5520	2	.1372
Reduced[b] Model (sequential SS)	Hour	1	70.9089	1	223.2020
	Hour^2	1	1.3339	1	2.8840
	Residual	143	.0284	121	.1316
MSE Reduced[a]-Complete[c]		2	.14046	2	.7217
F		2,143	**	2,121	**
	Chamber	1	.000288	1	<.0001
	Cow(chamber)	2	.000061	2	.0001
	Period	1	.000313$^+$	1	<.0001
	Treatment	1	.000181$^+$	1	<.0001
	Feed Intake[d]				
	Residual	2	.000019	2	

$^+$ P<.10

** P<.01

[a] One regression line fit to both treatments.

[b] Cow, period and treatment are absorbed in reduced model.

[c] Separate regression lines fit to each treatment.

[d] Feed intake as covariate was not statistically significant.

APPENDIX C
REGRESSION EQUATIONS FOR FIGURES IN CHAPTERS IV AND V

Table C-1. Regression equations for figures in Chapter IV of environmental treatments in chamber experiment where variables were measured over 26 hours.

Respiration Rate

Thermoneutral = $46.7 - 4.327x_1 + .82637589x_2 - .06768254x_3 + .00248377x_4 - 3.339572x10^{-5}\ x_5$

Heat Stress = $27.7 + 71.595x_1 - 14.84877881x_2 + 1.19867204x_3 - .04344659x_4 + .00059372x_5$

Rectal Temperature

Thermoneutral = $38.41 + 0.98x_1 - .00109989x_2 - .00120343x_3 + 7.4186003x10^{-5}\ x_4 - 1.3138606x10^{-6}\ x_5$

Heat Stress = $37.54 + 1.852x_1 - .32457007x_2 + .02236775x_3 - .00069895x_4 + 8.3980962x10^{-6}\ x_5$

Blood pH

Thermoneutral = $7.484 - .0159x_1 + .0027258x_2 - .00024163x_3 + 1.0260498x10^{-5}\ x_4 - 1.632185x10^{-7}\ x_5$

Heat Stress = $7.334 + .1191x_1 - .0240497x_2 + .00189647x_3 - 6.6096089x10^{-5}\ x_4 + 8.5575601x10^{-7}\ x_5$

Blood pCO_2

Thermoneutral = $37.6 - .671x_1 + .22913569x_2 - .03400139x_3 + .00183758x_4 - 3.2369304x10^{-5}\ x_5$

Heat Stress = $44.1 - 9.517x_1 + 1.87086514x_2 - .15095632x_3 + .00544692x_4 - 7.310552x10^{-5}\ x_5$

Blood HCO_3

Thermoneutral = $24.9 + 1.0357x_1 - .24977117x_2 + .01568705x_3 - .00030116x_4$

Heat Stress = $24.1 - .59997x_1 + .03971619x_2 - .00117827x_3 + 1.294292x10^{-5}\ x_5$

Urine pH

$$\text{Thermoneutral} = 7.419 + .1112x_1 - .02091825x_2 + .00127044x_3 - 2.5696133\times10^{-5}\ x_4$$

$$\text{Heat Stress} = 6.342 + .7891x_1 - .12544764x_2 + .00639645x_3 - .00010637x_4$$

Urine NH_4/Urine Creatinine

$$\text{Thermoneutral} = .0188 + .00063x_1 - 6.5233575\times10^{-6}\ x_2$$

$$\text{Heat Stress} = .0215 - .00359x_1 + .00025606x_2$$

Blood pO_2

$$\text{Thermoneutral} = 105 - 7.34x_1 + 1.88067552x_2 - .18761233x_3 + .00791473x_4 - .00011878x_5$$

$$\text{Heat Stress} = 102 - 8.53x_1 + 1.76390381x_2 - .15592827x_3 + .00640445x_4 - 9.8177899\times10^{-5}\ x_5$$

Ruminal pH

$$\text{Thermoneutral} = 5.94 + .068x_1 - .01645441x_2 + .00115639x_3 - 2.3611774\times10^{-5}\ x_4$$

$$\text{Heat Stress} = 5.51 + .316x_1 - .04215595x_2 + .00199767x_3 - 3.1555101\times10^{-5}\ x_4$$

Table C-2. Regression equations for figures in Chapter IV of environmental treatments in shade/no shade experiment where variables were measured over 26 hours.

Respiration Rate

Shade = $57.2 + 40.874x_1 - 8.79519699x_2 + .76175913x_3 - .02940086x_4 + .00041979x_5$

No Shade = $64.6 + 63.412x_1 - 15.14800088x_2 + 1.37052736x_3 - .05438732x_4 + .00079514x_5$

Rectal Temperature

Shade = $38.1 + 1.055x_1 - .19398563x_2 + .01581286x_3 - .00060451x_4 + 8.6959387x10^{-6}$

No Shade = $37.1 + 2.471x_1 - .49566561x_2 + .04047141x_3 - .00150135x_4 + 2.092408x10^{-5}$

Blood pH

Shade and No Shade Pooled = $7.364 + .0339x_1 - .0045183x_2 + 1.256067x10^{-4}\ x_3$

Blood pCO_2

Shade = $45.7 - 7.139x_1 + 1.3796819x_2 - .11146152x_3 + .00411450x_4 - 5.705438x10^{-5}\ x_5$

No Shade = $47.4 - 13.621x_1 + 2.8852618x_2 - .23864231x_3 + .00870969x_4 - .00011737x_5$

Blood HCO_3

Shade = $26.4 - 3.202x_1 + .637103024x_2 - .05635230x_3 + .00224908x_4 - 3.283512x10^{-5}\ x_5$

No Shade = $25.2 - 4.360x_1 + .9327777x_2 - .08139326x_3 + .00312046x_4 - 4.3509122x10^{-5}\ x_5$

Urine pH

$$\text{Shade} = 6.32 - .0073x_1 + .00038448x_2$$

$$\text{No Shade} = 7.81 - .2436x_1 + .00664024x_2$$

Hematocrit

$$\text{Shade} = 31.0 - 1.541x_1 + .32329892x_2 - .02934705x_3 + .00115508x_4 - 1.6336759\times10^{-5}\ x_5$$

$$\text{No Shade} = 27.5 + .635x_1 - .05157963x_2 - .00150698x_3 + .00020607x_4 - 4.176145\times10^{-6}\ x_5$$

Plasma Protein

$$\text{Shade} = 7.42 + .185x_1 - .03257545x_2 + .00186685x_3 - 3.3358155\times10^{-5}\ x_4$$

$$\text{No Shade} = 6.92 + .299x_1 - .04999492x_2 + .00276077x_3 - 4.9217577\times10^{-5}\ x_4$$

Table C-3. Regression equations for figures in Chapter V of dietary treatments in chamber experiment where variables were measured over 26 hours.

Respiration Rate

Basal $= 25.3 + 52.191x_1 - 11.25298704x_2 + .93190072x_3 - .03443451x_4 + .00047716x_5$

High Mineral $= 34.7 + 35.732x_1 - 7.23869065x_2 + .56155974x_3 - .01925392x_4 + .00024584x_5$

Rectal Temperature

Basal $= 37.6 + 1.420x_1 - .24254665x_2 + .01593064x_3 - .00046007x_4 + 5.033574x10^{-6}\ x_5$

High Mineral $= 37.2 + 1.902x_1 - .34566279x_2 + .02551034x_3 - .00085433x_4 + 1.0828992x10^{-5}\ x_5$

Urine pH

Basal $= 7.83 + .074x_1 - .01017286x_2 + .00028907x_3$

High Mineral $= 7.42 + .102x_1 - .01506289x_2 + .00041025x_3$

BIOGRAPHICAL SKETCH

Paul Lawrence Schneider was born in Bangor, Maine, on December 6, 1950. He received his elementary education in Bangor public schools. He attended Yeshiva University High School in New York City and graduated in 1968.

He entered Yeshiva University in September, 1968, and studied there for 2 years. He attended Tel Aviv University in Israel for his junior year and finished his senior year at City College of New York receiving a Bachelor of Arts degree in psychology.

In September, 1972, he returned to Israel and spent two years on a kibbutz working with the dairy herd.

He received a Bachelor of Science from the University of Maine in 1978 and a Masters of Science degree in 1980.

Presently he is a candidate for the degree of Doctor of Philosophy in animal science at the University of Florida.

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

David K. Beede, Chairman
Associate Professor of Dairy Science

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Charles J. Wilcox
Professor of Dairy Science

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Robert J. Collier
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I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

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I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

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